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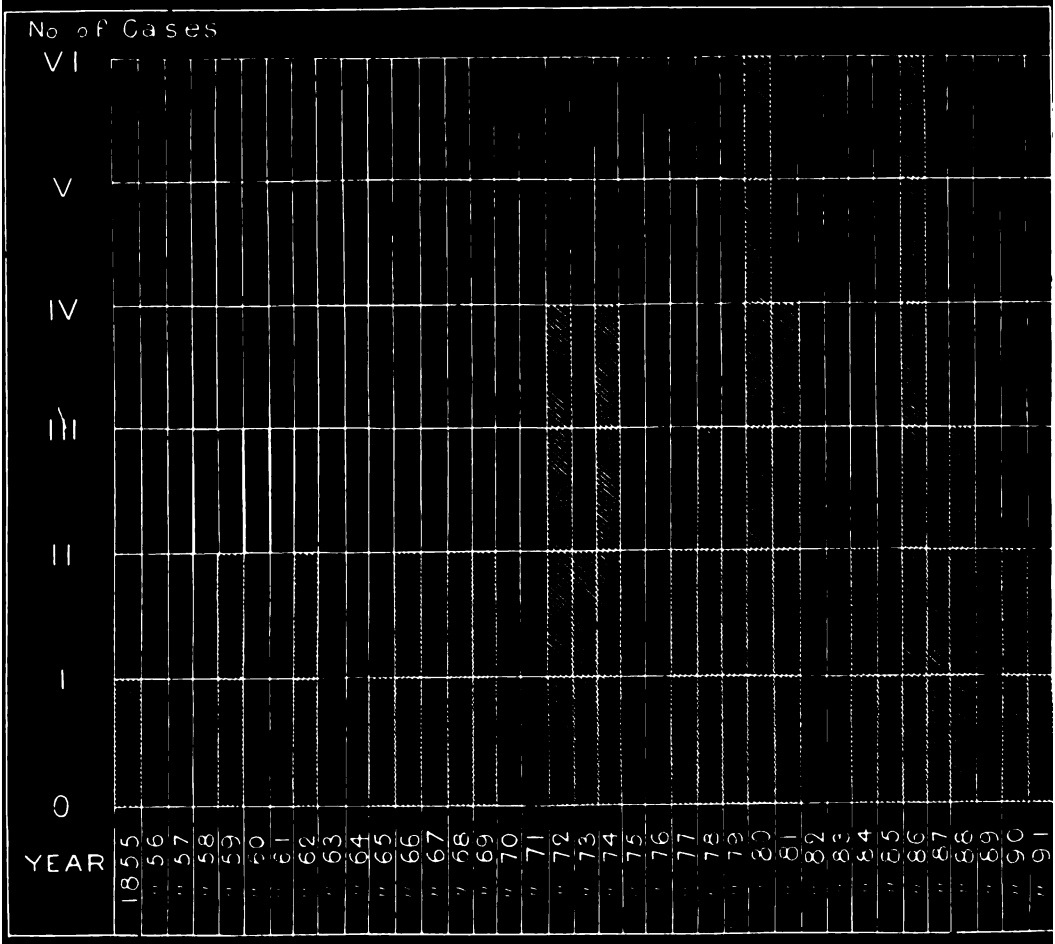

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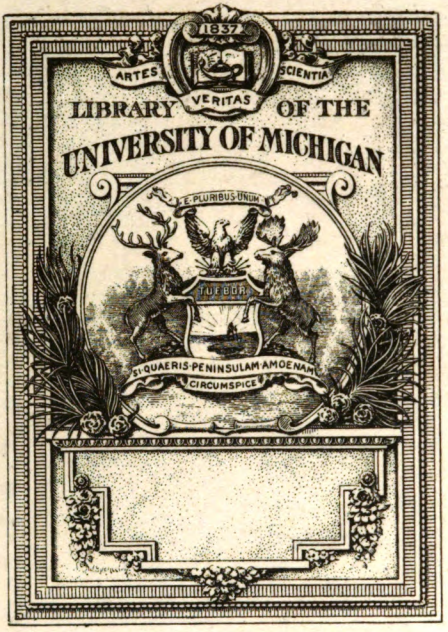

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PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON

VOLUME THE SEVENTY-FIFTH

(SECOND SERIES VOLUME THE FIFTY-SEVENTH)

LONDON

LONGMANS, GREEN AND CO.

PATERNOSTER ROW

1892

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Issued from the Society's House at 20, *Hanover Square, W.*

December, 1892.

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE.

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1835. HENRY EARLE
1837. RICHARD BRIGHT, M.D., D.C.L.
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* From 1805 to 1834 the Society was known as the Medico-Chirurgical Society of London.

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1887 FLOWER, SIR WILLIAM HENRY, K.C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.

1887 FOSTER, MICHAEL, LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Camp, Sunningdale.

1868 HUXLEY, the Right Hon. THOMAS HENRY, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, and Dresden.

1878 LUBBOCK, the Right Hon. SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Farnborough, Kent.

1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., Foreign Associate of the Academy of Sciences of France; Sheen Lodge, East Sheen, Mortlake.

1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D., F.R.S., M.P., Lucasian Professor of Mathematics in the University of Cambridge; Lensfield Cottage, Cambridge.

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1887 TURNER, SIR WILLIAM, D.C.L., LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh; 6, Eton Terrace, Edinburgh.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, and Geneva; Hindhead House, Hindhead, Surrey.

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1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna; 20, Alger Strasse, Vienna.

1883 CHARCOT, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Boulevard St. Germain, Paris.

1883 DUBOIS REYMOND, EMIL, M.D., Professor of Physiology, Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1887 VON ESMARCH, FRIEDRICH, M.D., Professor of Surgery in the University of Kiel.

1866 HANNOVER, ADOLPH, M.D., Copenhagen.

1873 VON HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics; Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France.

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L.—Hon. Librarian. C.—Member of Council.
Sci. Com.—A Scientific Committee. *Lib. Com.*—Library Committee.
Ho. Com.—House Committee. *Bldg. Com.*—Building Committee.

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OCTOBER, 1892.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

†1877 ABERCROMBIE, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 2, Henrietta street, Cavendish square.

*1851 ACLAND, SIR HENRY W., Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Oxford.

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1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton; 74, Brook street, Grosvenor square, W.

†1852 ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., LL.D.Glasgow, F.R.S., Regius Professor of Physic, Univ. Camb.; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square.

1890 ALLINGHAM, HERBERT WILLIAM, 25, Grosvenor street, Grosvenor square.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.

1881 ANDERSON, JAMES, A.M., M.D., Assistant Physician to the London Hospital and to the National Hospital for the Paralysed and Epileptic; 41, Wimpole street, Cavendish square.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; 9, Harley street, Cavendish square.

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1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

1891 ANDREWES, FREDERICK WILLIAM, M.B., 15, Upper Brook street.

1891 ANDREWS, LAUNCELOT, M.B., 21, Cheyne gardens, Manor street, Chelsea.

*1880 APPLETON, HENRY, M.D., 22, Lower Seymour street, Portman square.

1888 ARKLE, CHARLES, M.B., 66, Wimpole street, Cavendish square.

*1851 BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; The Bracken, Augustus road, Edgbaston.

1891 BAKER, CHARLES ERNEST, M.B., Royal Orthopædic Hospital; 15, Hanover square.

*1873 BAKER, J. WRIGHT, Consulting Surgeon to the Derbyshire General Infirmary; 101, Friar gate, Derby.

1865 BAKER, WILLIAM MORRANT, late Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1886-8. *Lib. Com.* 1876-7. *Trans.* 7.

1891 BALGARNIE, WILFRED, M.B., 47, South Molton street.

1887 BALL, JAMES BARRY, M.D., 54, Wimpole street, Cavendish square.

1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square, &c.; 56, Harley street, Cavendish square. *Trans.* 1.

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†1848 BALLARD, EDWARD, M.D., F.R.S., late Inspector, Medical Department, Local Government Board; 20, Curzon park, Chester. C. 1872. V.P. 1875-6. *Sci. Com.* 1889—. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

*1866 BANKS, SIR JOHN, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to Sir Patrick Dunn's and City of Dublin Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Royal University in Ireland; 45, Merrion square, Dublin.

1886 BANKS, WILLIAM MITCHELL, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

1879 BARKER, ARTHUR EDWARD JAMES, Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. *Trans.* 7.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1876 BARLOW, THOMAS, M.D., B.S., *Trustee for Debenture-holders*, Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892—. *Trans.* 2.

*1881 BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

†1861 BARNES, ROBERT, M.D., Lyss, Hants. C. 1877-8. V.P. 1889-90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D.

1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital and to the Westminster Hospital; 37, Wimpole street, Cavendish square.

1840 BARROW, BENJAMIN, Consulting Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

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1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886—. *Trans.* 2.

1890 BATEMAN, WILLIAM A. F., Bridge House, Richmond, Surrey.

1891 BATTEN, FREDERICK E., M.A., M.B., B.C.Cantab., 15, Airlie gardens, Campden hill, Kensington.

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

*1860 BEALEY, ADAM, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. C. 1886-7. *Referee*, 1882-5, 1889—. *Lib. Com.* 1881-5.

1880 BEEVOR, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and the Great Northern Hospital; 33, Harley street, Cavendish square. *Trans.* 1.

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1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Dental Hospital of London; 17, George street, Hanover square.

1877 BENNETT, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. *Referee*, 1892. *Trans.* 4.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Cairo, Egypt.

1890 BERRY, DAVID ANDERSON, M.B., C.M., 117, Goldhawk Road.

†1845 BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, B.S., Demonstrator of Anatomy, St. Bartholomew's Hospital; Surgeon to, and Lecturer on Clinical Surgery at, the Royal Free Hospital; 60, Welbeck street, Cavendish square.

1872 BEVERLEY, MICHAEL, M.D., Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

*1865 BICKERSTETH, EDWARD ROBERT, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.

1892 BICKERSTETH, ROBERT ALEXANDER, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

†1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

†1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

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†1851 BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D.

1881 BISS, CECIL YATES, M.D., Senior Assistant Physician to, and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.

1865 BLANDFORD, GEORGE FIELDING, M.D., late Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.

1891 BOKENHAM, THOMAS JESSOPP, 9, Upper Wimpole street, Cavendish square.

†1846 BOSTOCK, JOHN ASHTON, C.B., *Hon. Treasurer;* Hon. Surgeon to H.M. the Queen; Deputy Surgeon-General; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. T. 1888-92. *Sci. Com.* 1867.

1890 BOSTOCK, R. ASHTON, Surgeon, Scots Guards, 73, Onslow gardens, Brompton.

1869 BOURNE, WALTER, M.D. (Travelling) [care of the National Bank of India, 80, King William street, City].

1882 BOWLBY, ANTHONY ALFRED, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 4.

*1870 BOWLES, ROBERT LEAMON, M.D., 16, Upper Brook street, Grosvenor square. *Trans.* 1.

1886 BOXALL, ROBERT, M.D., Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 29, Weymouth street, Portland place.

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1884 BOYD, STANLEY, M.B., Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; 134, Harley street, Cavendish square.

1890 BRADFORD, JOHN ROSE, M.D., D.Sc., Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.

1874 BRADSHAW, A. F., C.B., Surgeon Major-General, Principal Medical Officer, H.M.'s Forces in India; Simla, India. [Agents: Holt & Co., 17, Whitehall place.]

1883 BRADSHAW, JAMES DIXON, M.B., 30, George street, Hanover square.

*1867 BRETT, ALFRED T., M.D., Watford House, Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, THOMAS, M.B., LL.D., Harrow-on-the-Hill, Middlesex.

1891 BRIGHT, ARCHIBALD LEONARD, 15, Margaret street, Cavendish square.

1890 BRINTON, ROLAND DANVERS, M.D., 8, Queen's Gate terrace.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Physician in Ordinary to H.R.H. the Prince of Wales; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891—. *Trans.* 5.

†1851 BRODHURST, BERNARD EDWARD, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

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1891 BRODIE, CHARLES GORDON, Senior Demonstrator of Anatomy, Middlesex Hospital Medical School; Assistant Surgeon, North-West London Hospital; 30, Harley street, Cavendish square.

1892 BRONNER, ADOLPH, M.D., 33, Manor row, Bradford.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Member of the Institut de France (Academy of Sciences); Professor of Medicine at the College of France. *Sci. Com.* 1862.

1888 BROWNE, HENRY LANGLEY, Moor House, West Bromwich.

1878 BROWNE, SIR JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; Queen Anne's Mansions, St. James's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 7, Norland place, Holland Park.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 70, Harley street. C. 1892—. *Sci. Com.* 1889—. *Referee,* 1886-91. *Lib. Com.* 1888-91. *Trans.* 2.

1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee,* 1880-87. *Lib. Com.* 1882-7.

1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee,* 1882-4. *Lib. Com.* 1868-71. *Trans.* 12. *Pro.* 1.

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1864 BUCHANAN, SIR GEORGE, M.D., F.R.S., late Medical Officer of the Local Government Board; Member of the Senate of the University of London; 27, Woburn square.

1864 BUCKLE, FLEETWOOD, M.D.

1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 35, Clarges street, Piccadilly.

1881 BULLER, AUDLEY CECIL, M.D.

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

1873 BUTLIN, HENRY TRENTHAM, Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887–8. *Trans.* 3.

1871 BUTT, WILLIAM F.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the Hospital for Paralysis and Epilepsy, Queen's square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

*1851 CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1890 CAGNEY, JAMES, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 93, Wimpole street, Cavendish square. *Trans.* 1.

1885 CAHILL, JOHN, 12, Seville street, Lowndes square.

1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.

1891 CAMPBELL, HENRY JOHNSTONE, M.D., Senior Demonstrator of Biology and Demonstrator of Physiology, Guy's Hospital; Assistant Physician, East London Children's Hospital; 10, St. Thomas's street, Southwark.

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1888 CARLESS, ALBERT, M.B., M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1888 CARTER, WILLIAM JEFFREYS BECHER, Aliwal North, Cape Colony.

1879 CARTWRIGHT, S. HAMILTON.

1888 CAUTLEY, EDMUND, M.B., B.C., 15, Upper Brook street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888—. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Consulting Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1884 CHAFFEY, WAYLAND CHARLES, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.D., Physician Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. *Referee*, 1891—. *Lib. Com.* 1885—. *Trans.* 7.

1859 CHANCE, FRANK, M.B., Burleigh House, Sydenham Hill.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; Albert Mansions, 106, Victoria street, Westminster.

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*1881 CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital; 22, Temple row, Birmingham. *Trans.* 3.

1868 CHEADLE, WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889—. *Bldg. Com.* 1889-92. *Referee,* 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, M.B., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 59, Welbeck street, Cavendish square. *Lib. Com.* 1886-8, 1891—. *Trans.* 1.

1890 CHILDS, CHRISTOPHER, M.D., 2, Royal terrace, Weymouth.

*1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Central Hospital; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee,* 1873-80.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892—. *Referee,* 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., *President, Trustee,* Consulting Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875. V.P. 1888. P. 1892.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1892 CLARK, JAMES CHARLES, Croft House, Margate road, Southsea.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Surgeon and Ophthalmic Surgeon to the Miller Hospital; 41, Lee terrace, Blackheath, and 112, Harley street.

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1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist, St. Mary's Hospital, Paddington.

†1848 CLARKE, JOHN, M.D. C. 1866.

1888 CLARKE, ROBERT HENRY, M.B., Westwood, Isle of Thanet, Kent.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital; 46, Harley street, Cavendish square.

†1879 CLUTTON, HENRY HUGH, M.A., M.B., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Victoria Hospital for Children; 2, Portland place.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.

1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester square.

1868 COCKLE, JOHN, A.M., M.D., F.L.S., Consulting Physician to the Royal Free Hospital; 5, Suffolk place, Pall Mall. *Trans.* 2.

1891 COOK, HERBERT GEORGE, M.B., B.S., 162, Cromwell road, South Kensington.

1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1868 CORNISH, WILLIAM ROBERT, C.I.E., late Surgeon-General to the Government of Madras; Hon. Physician to H.M. the Queen; 8, Cresswell gardens, The Boltons.

*1860 CORRY, THOMAS CHARLES STEUART, M.D., Ormeau terrace, and 1, Glenfield place, Belfast.

1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park.

1890 COTES, CHARLES EDWARD HENRY, M.B., 20, Wilton street, Grosvenor place.

1892 COTTERELL, EDWARD, 5, West Halkin street, Belgrave square.

1891 COUMBE, JOHN BATTEN, M.D., Wargrave, Henley-on-Thames.

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†1860 COUPER, JOHN, Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Joint Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. *Referee*, 1892—.

†1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., M.Ch., LL.D., Hon. Surgeon to the Queen; Director-General, Army Medical Department (Retired); 5, St. John's Park, Blackheath. C. 1887.

*1869 CRESSWELL, PEARSON R., Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; late Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.

1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.

1892 CROSS, FRANCIS RICHARDSON, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

1872 CROSSE, THOMAS WILLIAM, Consulting Surgeon to the Norfolk and Norwich Hospital; 45, St. Giles's street, Norwich.

1890 CROWLE, THOMAS HENRY RICKARD, 3, Campden Hill road, Kensington.

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1888 Cullingworth, Charles James, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 46, Brook street, Grosvenor square.

1879 Cumberbatch, A. Elkin, Aural Surgeon to St. Bartholomew's Hospital, and National Hospital for the Paralysed and Epileptic; 17, Queen Anne street, Cavendish square.

1873 Curnow, John, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 3, George street, Hanover square. *Referee*, 1884—.

1886 Dakin, William Radford, M.D., Obstetric Physician to St. George's Hospital; 57, Welbeck street, Cavendish square.

1872 Dalby, Sir William Bartlett, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. *Trans.* 3.

1884 Dallaway, Dennis, 5, Duchess street, Portland place.

1891 Dalton, Norman, M.D., 4, Mansfield street, Cavendish square.

1877 Darbishire, Samuel Dukinfield, M.D., Physician to the Radcliffe Infirmary, Oxford; Plas Mawr, Penmaenmawr, N. Wales.

1879 Darwin, Francis, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.

1874 Davidson, Alexander, M.D., Physician to the Liverpool Royal Infirmary; 2, Gambier terrace, Liverpool.

1852 Davies, William, M.D., 2, Marlborough buildings, Bath.

1876 Davies-Colley, J. Neville C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892—. *Referee*, 1890-91. *Trans.* 2.

1878 Davy, Richard, Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.

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*1882 DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1889 DEAN, HENRY PERCY, M.B., M.S., 84, Wimpole street, Cavendish square.

1889 DELÉPINE, SHERIDAN, B.S., M.B., Professor of Pathology, Owens College, Manchester. *Trans.* 1.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 4.

†1859 DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889—. *Trans.* 14.

†1891 DICKINSON, WILLIAM LEE, M.B., 9, Chesterfield street, Mayfair.

†1839 DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

1889 DODD, HENRY WORK, Assistant Surgeon to the Royal Free Hospital, and to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.

1888 DONELAN, JAMES, M.B., M.C., 2, Upper Wimpole street, Cavendish square.

1879 DONKIN, HORATIO BRYAN, M.B.Oxon., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

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1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Lib. Com.* 1891—. *Trans.* 1.

1891 DOVE, P. W., St. Bartholomew's Hospital.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890-91. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1884 DRAGE, LOVELL, M.A., M.B., B.S.Oxon., Burleigh Mead, Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.

1885 DRUMMOND, DAVID, M.D., 7, Saville place, Newcastle-on-Tyne.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Senior Physician to the Metropolitan Hospital; 23, Sackville street, Piccadilly.

†1865 DUCKWORTH, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885—. *Trans.* 2.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

1871 DUKE, BENJAMIN, Windmill House, Clapham Common.

*1871 DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, MAJOR CHARLES, M.D., 6, Wellesley Villa, Wellesley road, Croydon.

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1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

*1889 DUNCAN, JOHN, M.D., St. Petersburg, Russia.

1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon and Pathologist at the West London Hospital; 39, Welbeck street, Cavendish square.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V.P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1887 EASMON, JOHN FARRELL, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester place, Hyde Park. C. 1892—.

1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.

1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.

1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 55, Harley street, Cavendish square.

1824 EDWARDS, GEORGE.

1891 ELAM, GEORGE, 75, Harley street, Cavendish square.

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1887 ELLIOTT, JOHN, Whitefriars Lodge, Chester.

1848 ELLIS, GEORGE VINER, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

*1854 ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1889 ELLISTON, WILLIAM ALFRED, M.D., Stoke Hall, Ipswich.

†1842 ERICHSEN, JOHN ERIC, LL.D., F.R.S., M.Ch., Surgeon Extraordinary to H.M. the Queen; President of, and Emeritus Professor of Surgery in, University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-8, 1884-89. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to Out-Patients at the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. *Sci. Com.* 1889—. *Trans.* 1.

*1875 FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 19, Great Victoria street, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 59, Warrior square, St. Leonard's-on-Sea.

1862 FARQUHARSON, ROBERT, M.D., LL.D., M.P., Migvie Lodge, Porchester gardens, Hyde Park (Finzean, Aboyne, Aberdeenshire). *Lib. Com.* 1876-80.

1872 FAYRER, SIR JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council; President of the Medical Board at the India Office; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.

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1887 FEENY, MICHAEL HENRY, Les Avants, Montreux, Switzerland.

*1872 FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882—. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuropathology in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891—. *Trans.* 2.

1852 *FIELD, ALFRED GEORGE.

1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.

1879 FINLAY, DAVID WHITE, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; Aberdeen. *Referee*, 1891—. *Trans.* 2.

1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park.

1891 FLETCHER, HERBERT MORLEY, M.B., 98, Harley street, Cavendish square.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

*1864 FOLKER, WILLIAM HENRY, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 DE FONMARTIN, HENRY, M.D., 1, Anchor Gate terrace, Portsea, Hants.

1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.

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1865 FOSTER, SIR BALTHAZAR WALTER, M.D., M.P., Emeritus Professor of Medicine at the Queen's College, Birmingham, and Consulting Physician to the Birmingham General Hospital; 55, Temple row, Birmingham, and 11, George street, Hanover square, London.

1892 FOSTER, MICHAEL GEORGE, M.A., M.B., Great Shelford, Cambridge.

1882 FOWLER, JAMES KINGSTON, M.A., M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1887 FOX, RICHARD HINGSTON, M.D., Physician to St. Luke's Mission Dispensary; 23, Finsbury square.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Skin Diseases to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1871 FRANK, PHILIP, M.D., Cannes, France.

*1884 FRANKS, KENDAL, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; Surgeon in Ordinary to the Lord Lieutenant; 6, Fitzwilliam square, Dublin. *Trans.* 1.

1843 FRASER, PATRICK, M.D. C. 1866.

*1889 FREEMAN, HENRY WILLIAM, 24, The Circus, Bath.

1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.

1876 FURNER, WILLOUGHBY, M.D., Surgeon to the Sussex County Hospital; 13, Brunswick square, Brighton.

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*1864 GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

†1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892—. *Referee*, 1882-91. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

1885 GAMGEE, ARTHUR, M.D., F.R.S., Davos, Switzerland.

1865 GANT, FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886—. *Lib. Com.* 1882-5. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

†1854 GARROD, SIR ALFRED BARING, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1886 GARROD, ARCHIBALD EDWARD, M.A., M.D., Assistant Physician to the West London Hospital; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 5.

1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.

*1889 GASKELL, WALTER HOLBROOK, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; Petersfield House, Parkside, Cambridge.

1887 GAY, JOHN, 119, Upper Richmond road, Putney.

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1866 GEE, SAMUEL JONES, M.D., *Hon. Librarian, Chairman of Trustees for Debenture-holders*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887-92. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

1885 GELL, HENRY WILLINGHAM, M.B., 43, Albion street, Hyde Park.

1878 GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee*, 1884—. *Trans.* 1.

1884 GIBBES, HENEAGE, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.

1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Secretary*, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London, and Surgeon to the Hospital for Consumption, Brompton; Consulting Surgeon to the North-Eastern Hospital for Children; 19, Wimpole street, Cavendish square. S. 1892—. *Referee*, 1886-91. *Trans.* 7.

†1870 GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Senior Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 12, Queen Anne street, Cavendish square. *Trans.* 1.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

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1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital; Surgeon to St. Mark's Hospital; 17, Devonshire place, Upper Wimpole street.

*1890 GORDON, WILLIAM, M.B., Barnfield Lodge, Exeter.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892—. *Lib. Com.* 1891. *Trans.* 2.

1891 GOW, WILLIAM J., M.D., 13, Upper Wimpole street, Cavendish square.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

†1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street, Cavendish square.

1868 GREEN, T. HENRY, M.D., Physician to Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.

1889 GREENE, GEORGE EDWARD JOSEPH, Monte Vista, Ferns, County Wexford.

*1875 GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1882 GRESSWELL, DAN ASTLEY, M.A., M.D., D.P.H., Melbourne, Victoria.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St. Bartholomew's Hospital; University Lecturer and Examiner in Obstetrics, Cambridge; 114, Harley street, Cavendish square.

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1889 GRIFFITHS, JOSEPH, M.A., M.D., C.M., Assistant to the Professor of Surgery in the University of Cambridge; 17, Fitzwilliam street, Cambridge.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Obstetric Physician to the Out-patients at the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Pitt House, 15, Johnstown street, Bath.

1889 GUBB, ALFRED S, M.D.Paris; 29, Gower street.

1883 GUNN, ROBERT MARCUS, M.A., M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street, and to the National Hospital for the Paralysed and Epileptic; Assistant Ophthalmic Surgeon to University College Hospital; 54, Queen Anne street, Cavendish square.

1890 GUTHRIE, LEONARD GEORGE, M.B., B.S., Physician to the Regent's Park Hospital for Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician and Pathologist to the Children's Hospital, Paddington Green; 24, Upper George street, Bryanston square.

1886 HABERSHON, SAMUEL HERBERT, M.D., 70, Brook street, Grosvenor square.

1888 HADDEN, WALTER BAUGH, M.D., Assistant Physician and Lecturer on Materia Medica and Therapeutics, and Joint Lecturer on Pathological Anatomy at St. Thomas's Hospital; Assistant Physician, Hospital for Sick Children; 21, Welbeck street, Cavendish square.

1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 5.

1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

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1881 HALL, FRANCIS DE HAVILLAND, M.D., Physician to Out-patients and to the Throat Department at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square.

1891 HAMER, WILLIAM HEATON, M.D., 73, Dartmouth Park Hill, Highgate.

1870 HAMILTON, ROBERT, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magheraybuoy, Portrush, Co. Antrim, Ireland.

1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.

†1856 HARE, CHARLES JOHN, M.D., *Hon. Treasurer*, Consulting Physician to University College Hospital, and late Professor of Clinical Medicine in University College, London; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887—. *Bldg. Com.* 1889-92.

†1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. V.P. 1891-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3, 1866, 1889—. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882—. *Sci. Com.* 1879. *Trans.* 10.

1892 HAROLD, JOHN, 72, Wimpole street, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. *Trans.* 1.

1892 HARSANT, WILLIAM HENRY, 16, Pembroke road, Clifton, Bristol.

1854 HAVILAND, ALFRED.

1890 HAVILAND, FRANK PAPILLON, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.

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1870 HAWARD, J. WARRINGTON, *Trustee for Debenture-holders*, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. *Lib. Com.* 1881-4. *Sci. Com.* 1889—. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

1885 HAWKINS, FRANCIS HENRY, M.B., 59, Wimpole street, Cavendish square.

1891 HAWKINS, HERBERT PENNELL, M.B., B.C., Assistant Physician to St. Thomas's Hospital; 38, Weymouth street, Portland place.

†1848 HAWKSLEY, THOMAS, M.D., Beomands, Chertsey, Surrey.

1875 HAYES, THOMAS CRAWFORD, M.A., M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Lecturer in Practical Obstetrics in King's College; Physician for Diseases of Women to Royal Free Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1891 HAYWARD, JOHN ARTHUR, M.B.

1861 HAYWARD, WILLIAM HENRY.

†1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.

1882 HENSLEY, PHILIP JOHN., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

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1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.D., 13, Upper Wimpole street, Cavendish square. *Trans.* 1.

1887 HEWITT, FREDERIC WILLIAM, M.D., Instructor in, and Lecturer on Anæsthetics at, the London Hospital; Anæsthetist at the Dental Hospital of London; 10, George street, Hanover square. *Trans.* 1.

†1855 HEWITT, W. M. GRAILY, M.D., Emeritus Professor of Midwifery in University College, London, and Consulting Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877—. *Lib. Com.* 1868, 1874.

1880 HICKS, CHARLES CYRIL, M.D., Wokingham, Berks.

1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1890 HILL, G. WILLIAM, M.D., B.Sc., 24, Wimpole street, Cavendish square.

†1843 HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 HOLLAND, PHILIP ALEXANDER, M.A.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.D., Assistant Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

†1856 HOLMES, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg. Com. (Chairman)* 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892—. *Ho. Com.* 1892—. *Trans.* 8.

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†1846 HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington Gardens. C. 1862-3. V.P. 1879-80.

†1846 HOLTHOUSE, CARSTEN, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Assistant Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; 25, Cavendish square. *Trans.* 1.

1865 HOWARD, BENJAMIN, M.D. [New York, U.S.] *Trans.* 1.

1881 HOWARD, HENRY, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.

1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 2.

1886 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, Surgical Registrar, Middlesex Hospital; 6, Chandos street, Cavendish square.

†1857 HULKE, JOHN WHITAKER, F.R.S., *Hon. Librarian*, Surgeon to the Middlesex Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-92. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 9.

1889 HUMPHERY, FRANCIS WILLIAM, M.A., M.B., 63, Prince's gate.

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1855 HUMPHRY, SIR GEORGE MURRAY, M.D., D.Sc., LL.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 9.

1882 HUMPHRY, LAURENCE, M.D., 3, Trinity street, Cambridge.

1889 HUNTER, WILLIAM, M.D., 61, Wimpole street, Cavendish square.

1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital; 21, Norfolk crescent, Hyde Park.

1849 HUSSEY, EDWARD LAW, Coroner of the City of Oxford; 24, Winchester road, Oxford. *Trans.* 1.

†1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883—. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 1, Park crescent, W.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D.

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

†1841 JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.

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1883 JACOBSON, WALTER HAMILTON ACLAND, M.A., M.B., M.Ch., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Trans.* 2.

1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens.

*1883 JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

†1851 JENNER, SIR WILLIAM, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1884 JENNINGS, CHARLES EGERTON, M.S., M.B.

1881 JENNINGS, WILLIAM OSCAR, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Cancer Hospital, Brompton; 1, Buckingham Palace Mansions.

1883 JESSOP, WALTER H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

†1847 JOHNSON, SIR GEORGE, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; Emeritus Professor of Clinical Medicine; Fellow and Member of the Council of King's College, London; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10. *Pro.* 1.

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.

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1889 JOHNSON, HAROLD J., Senior Assistant, Gloucester County Asylum.

1889 JOHNSON, RAYMOND, M.B., B.S., Surgeon to Out-Patients at the Great Northern Central Hospital and the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.

1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

*1875 JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange.]

1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.

*1848 KENDELL, DANIEL BURTON, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.

*1890 KERR, J. G. DOUGLAS, M.B., C.M., 6, The Circus, Bath.

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1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital, Shaftesbury avenue, W.C.; 11, Harley street, Cavendish square.

*1877 KHORY, RUSTOMJEE NASERWANJEE, M.D., Honorary Obstetric Physician to the Bai Motlibari and Sir Dinsha Petil Hospitals, &c.; Hormazd Villa, Khumballa hill, Bombay.

†1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton, and Assistant Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.

†1851 KINGDON, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1883 KNAPTON, GEORGE, Northumberland House, Richmond, Surrey.

1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.

1889 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch.Oxon., 1 Northampton villas, Swansea.

1891 LANE, HUGH, 11, The Circus, Bath.

†1840 LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital, the West London Hospital, and to the Lock Hospital; "St. Mary's," Madeley road, Ealing. C. 1849-50. V.P. 1865. *Referee*, 1850.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to Guy's Hospital and to the Hospital for Sick Children; 8, St. Thomas's street, Southwark. *Trans.* 3.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885—. *Lib. Com.* 1879-80, 1888—. *Trans.* 2.

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*1873 LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.

1816 LAWRENCE, G. E.

1890 LAWRENCE, HENRY CRIPPS, 12, Sussex gardens, Hyde Park.

1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.

*1890 LAWRIE, EDWARD, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.

1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., C.M., Melbourne, Victoria, Australia.

1892 LAZARUS-BARLOW, WALTER SYDNEY, M.B., 16, Bryanston street, Portman square. *Sci. Com.* 1892—.

1892 LEADAM, WILLIAM WARD, M.D., 80, Gloucester terrace, Hyde Park.

*1886 LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary; 41, Lowther street, Carlisle.

1882 LEDWICH, EDWARD L'ESTRANGE, Anatomist to the Royal College of Surgeons, Ireland; 31, Harcourt street, Dublin.

†1843 LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1883 LEESON, JOHN RUDD, M.D., C.M., Clifden House, Twickenham.

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Elected

1869 Legg, John Wickham, M.D., C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1886 Lewers, Arthur Hamilton Nicholson, M.D., Obstetric Physician to the London Hospital; 60, Wimpole street, Cavendish square.

1872 Liebreich, Richard (Consulting Ophthalmic Surgeon to St. Thomas's Hospital, London); Paris.

1878 Lister, Sir Joseph, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery at King's College, London; and Senior Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.

*1872 Little, David, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Opthalmology at the Victoria University; 21, St. John street, Manchester.

1891 Little, Ernest Muirhead, 40, Seymour street, Portman square.

*1889 Little, James, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens' and the Children's Hospitals; 14, Stephen's Green North, Dublin.

1889 Little, John Fletcher, M.B., 60, Welbeck street, Cavendish square.

1871 Little, Louis Stromeyer, Shanghai, China.

1881 Lockwood, Charles Barrett, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Demonstrator of Operative Surgery at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. *Trans.* 2.

1860 Longmore, Sir Thomas, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff (Retired); Foreign Corresponding Member, "Académie de Médecine;" Assoc. Soc. Chir. de Paris; Officer of Legion of Honour; The Paddock, Woolston, Hants. *Trans.* 2.

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1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Belmont, Wateringbury, Kent.

1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Corresponding Member of the "Société de Chirurgie" of Paris; 18, Finsbury square. *Trans.* 2.

1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Out-patients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; Official Analyst to the Home Office; 47, Weymouth street, Portland place.

1883 LUND, EDWARD, Emeritus Professor of Surgery, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1887 LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hampstead.

1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.

1889 MACALISTER, DONALD, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

†1873 MACCARTHY, JEREMIAH, M.A., Surgeon to the London Hospital and Lecturer on Surgery at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.

1867 MAC CORMAC, SIR WILLIAM, M.A., M.Ch., D.Sc., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Referee*, 1889—. *Trans.* 1.

1887 MACDONALD, GEORGE CHILDS, M.D.

1866 MACGOWAN, ALEXANDER THORBURN, M.D.

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1880 McHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.

*1859 M'INTYRE, JOHN, M.D., LL.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.

1876 MACKEY, EDWARD, M.D., Senior Physician to the Royal Alexandra Hospital for Sick Children; Assistant Physician to the Sussex County Hospital; 1, Brunswick road, Hove, Brighton.

*1854 MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, The Cedars, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1889 MACLEHOSE, NORMAN MACMILLAN, M.B., C.M., 13, Queen Anne street, Cavendish square.

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90. *Lib. Com.* 1886-90.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

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1886 MAGUIRE, ROBERT, M.D., Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Assistant Physician to the Consumption Hospital, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. *Trans.* 1.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 1.

1891 MANBY, ALAN REEVE, M.D., Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.

1890 MANSON, PATRICK, M.D., C.M., LL.D., 21, Queen Anne street, Cavendish square.

1888 MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish square.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-2. *Lib. Com.* 1880-1. *Trans.* 4.

1892 MARTIN, CHRISTOPHER, M.B., C.M., 3, Crescent, Birmingham.

1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.

1884 MARTIN, SIDNEY HARRIS COX, M.D., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; 10, Mansfield street, Portland place.

1892 MASTERS, JOHN ALFRED, M.D., 35, Bruton street, Berkeley square, and Westall House, Brook Green.

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1883 MAUDSLEY, HENRY CARR, M.D., 22, Collins street, Melbourne, Victoria.

1892 MAUNSELL, HENRY WIDENHAM, M.A., M.D.Dublin, 37, Stanhope gardens, Queen's gate.

1891 MAY, WILLIAM PAGE, M.D., B.Sc., Goldburn, Eltham road, Blackheath.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1865 MEDWIN, AARON GEORGE, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square.

1891 MERCIER, CHARLES ARTHUR, M.B., 2, Henrietta street, Cavendish square.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 21, Manchester square. *Trans.* 1.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1882 MILLS, JOSEPH, 28, Queen Anne street, Cavendish square.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Beaumont Lodge, Worple road, Wimbledon.

1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.

1883 MONEY, ANGEL, M.D.

1873 MOORE, NORMAN, M.D., Assistant Physician and Lecturer on Pathological Anatomy at St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. *Referee*, 1886-90. *Sci. Com.* 1889—.

1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 2.

1891 MORRIS, GRAHAM, Wallington, Surrey.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Trans.* 10.

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1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—.

1885 MOTT, FREDERICK WALKER, M.D., Assistant Physician and Lecturer on Physiology, Charing Cross Hospital; 84, Wimpole street, Cavendish square.

†1888 MURRAY, HUBERT MONTAGUE, M.D., 27, Savile row.

1873 MURRAY, J. IVOR, M.D., 24, Huntriss row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Physician to Out-patients, and Lecturer on Materia Medica and Therapeutics at the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1892 MYDDELTON-GAVEY, E. HERBERT, 94, Wimpole street, Cavendish square.

1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., 2, Manchester square.

1889 NAPIER, FRANCIS HORATIO, M.B.

1881 NALL, SAMUEL, M.B.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee*, 1892—.

1889 NEVINS, ARTHUR EDWARD, Eastwood place, Hanley, Staffordshire.

†1843 NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Trenarren, Newquay, Cornwall.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

*1847 NOURSE, WILLIAM EDWARD CHARLES, Lorne House, Avenue road, Torquay.

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1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1884 OAKES, ARTHUR, M.D.

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; Greenhill Park, Harlesden.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, B.Sc., M.B., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1891 OGLE, CYRIL, M.A., M.B., 30, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.A., M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

*1855 OGLE, WILLIAM, M.A., M.D., late Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

*1883 OLIVER, THOMAS, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.

*1871 O'NEILL, WILLIAM, M.D., late Physician to the Lincoln Lunatic Hospital, 2, Lindum road, Lincoln.

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1892 OPENSHAW, T. HORROCKS, M.B., M.S., Assistant Surgeon to, and Senior Demonstrator of Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889–90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.

1890 ORD, WILLIAM WALLIS, M.D., 2, Queen street, Mayfair.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. *Trans.* 2.

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.

1892 PAGE, HARRY MARMADUKE, 107, London Wall.

1887 PAGET, CHARLES EDWARD, Medical Officer of Health for the County Borough of Salford; North Bentcliffe, Eccles, Lancashire.

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†1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 PAGET, STEPHEN, 57, Wimpole street, Cavendish square.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.

1887 PARDINGTON, GEORGE LUCAS, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1873 PARKER, ROBERT WILLIAM, Senior Surgeon to the East London Hospital for Children; Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. *Bldg. Com.* 1889-92. *Referee*, 1891—. *Lib. Com.* 1885-87, 1892—. *Ho. Com.* 1892—. *Trans.* 4.

1885 PARKER, RUSHTON, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1891 PARKIN, ALFRED, M.S., M.D., 5, Albion street, Hull. *Trans.* 1.

1889 PARSONS, J. INGLIS, M.D., Physician to Out-patients, Chelsea Hospital for Women; 3, Queen street, Mayfair.

1883 PASTEUR, WILLIAM, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.

1891 PATERSON, WILLIAM BROMFIELD, 64, Brook street, Grosvenor square.

1891 PATON, EDWARD PERCY, M.D., 103, Highbury quadrant.

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1865 PAVY, FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Laggan House, Maidenhead.

*1855 PEMBERTON, OLIVER, Consulting Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; H.M. Coroner, Birmingham; 65, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Teme, Worcester.

1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.

1890 PERRY, EDWIN COOPER, M.D., Assistant Physician and Demonstrator of Pathology at Guy's Hospital; The College, Guy's Hospital.

*1879 PESIKAKA, HORMASJI DOSABHAI, late Hon. Surgeon to the G. T. Hospital (Bombay); 43, Hornby Road, Bombay.

*1878 PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Palace Court, Notting hill gate.

1888 PHILLIPS, JOHN, M.A., M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square. *Trans.* 1.

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1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients and Lecturer on Materia Medica at St. Mary's Hospital, Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee*, 1882-3, 1891—. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.

1891 PIERCE, BEDFORD, M.D., The Retreat, York.

†1841 PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark. *Trans.* 1.

1889 PITTS, BERNARD, M.B., M.C., Assistant Surgeon to St. Thomas's Hospital; 31, Harley street, Cavendish square.

1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, Assistant Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

†1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

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1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street, Cavendish square. C. 1890-91. *Referee* 1887-89, 1892—. *Trans.* 2.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1892 POWELL, HERBERT ANDREWS, M.A., M.D., M.Ch., 9, St. Thomas's street, Winchester.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption and Diseases of the Chest at Brompton; 62, Wimpole street, Cavendish square. S. (Oct.), 1883-5. C. 1887-8. *Referee* 1879-83, 1886. *Trans.* 3.

1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Surgery at St. Bartholomew's Hospital; Surgeon to Out-patients at Victoria Hospital for Children; 26, Bloomsbury square. *Trans.* 1.

1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892—. *Referee*, 1870-81, 1891—. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.

1857 †PRIESTLEY, WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to, Lecturer on Practical Medicine, and Physician in Charge of Skin Department at, the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 1.

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1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. *Lib. Com.* 1887—. *Trans.* 1.

†1850 QUAIN, SIR RICHARD, Bart., M.D., (Hon.) M.D.Dublin, LL.D.Ed., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton, and to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. C. 1889. *Referee*, 1885-8.

1857 VON RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1890 RANSOM, WILLIAM BRAMWELL, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1892 RAYNER, HENRY, M.D., 2, Harley street, Cavendish square.

1891 READ, HENRY GEORGE, 30, Finsbury square, and "Martins," Shipbourne, Kent.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1891 REECE, RICHARD JAMES, 34, Eardley crescent, South Kensington.

1882 REID, JAMES, M.D., C.B., Resident Physician in Ordinary to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

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Elected

1891 REMFRY, LEONARD, M.D., 60, Great Cumberland place.

1891 RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 44, Lancaster gate.

†1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Emeritus Professor of Medicine in University College; Consulting Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.

1887 RICHARDSON, GILBERT, M.A., M.D., Hawthorn House, Putney.

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889—. *Trans.* 6.

1889 RIVERS, W. H. RIVERS, M.D., Bethlem Hospital, St. George's road, S.E.

1871 RIVINGTON, WALTER, M.S., Consulting Surgeon to the London Hospital; 95, Wimpole street, Cavendish square. C. 1885-6. *Trans.* 4.

*1871 ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester; 11, St. John street, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics, and of Clinical Medicine, in University College, London; and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. *Sci. Com.* 1889—.

1889 ROBERTS, H. LESLIE, M.B., C.M., 31, Rodney street, Liverpool.

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1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

*1888 ROBINSON, FREDERICK WILLIAM, M.D., C.M., Huddersfield.

1889 ROBSON, ARTHUR WILLIAM MAYO, Hillary place, Leeds. *Trans.* 2.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1890 ROLLESTON, HUMPHRY DAVY, M.A., M.D., Pathologist and Lecturer on Pathology at St. George's Hospital; 13, Upper Wimpole street, Cavendish square.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.

†1857 ROSE, HENRY COOPER, M.D., Consulting Surgeon to the Hampstead Dispensary; 53, Rosslyn hill, Hampstead. C. 1886-7. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery at King's College; Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.

1889 ROSS, DANIEL MCCLURE, 54, Upper Berkeley street.

1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Warden of the College of St. Mary's Hospital; Assistant Surgeon to the Royal Free Hospital; 33, Westbourne terrace, Hyde Park. *Trans.* 1.

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1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Obstetric Physician to Out-patients, and Lecturer on Practical Midwifery at the Charing Cross Hospital; 14A, Manchester square.

†1849 ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1891 RUFFER, MARC ARMAND, M.A., M.D., 19, Iddesleigh Mansions, Westminster.

1891 RUSSELL, J. S. RISIEN, M.B., C.M., 4, Queen Anne street, Cavendish square.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital and Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

†1855 SANDERSON, JOHN BURDON, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

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1867 SANDFORD, FOLLIOTT JAMES, M.D., Surgeon-Major; Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician and Vice-President, North-Eastern Hospital for Children; 84, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889—. *Trans.* 2.

1891 SANTI, PHILIP ROBERT WILLIAM, 30, Clanricarde gardens.

1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason College; 83A, Edmund street, Birmingham.

†1845 SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; 13A, George street, Hanover square. C. 1872-3.

1891 SAUNDERS, FREDERICK WILLIAM, M.B., B.C., 17, Barkston gardens, South Kensington.

1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.

1859 SAVORY, SIR WILLIAM, Bart., F.R.S., Surgeon Extraordinary to H.M. the Queen; Consulting Surgeon to St. Bartholomew's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.

1892 SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.Ph., 11, Portland place.

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1887 SCOTT, HARRY, M.D., 47, St. Ermin's mansions, Westminster.

*1861 SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1892 SEGUNDO, CHARLES SEMPILL DE, 2, Aldridge road villas, Westbourne park.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Artwood road, S.W.

1877 SEMON, FELIX, M.D., Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Trans.* 1.

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician and Joint Lecturer on Pathology at St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1886 SHAW, LAURISTON ELGIE, M.D., Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., Assistant Surgeon, Charing Cross Hospital; 20, Stratford place, Oxford street. *Trans.* 3.

†1859 SIBLEY, SEPTIMUS WILLIAM, The Hermitage, Bletchingley, Surrey. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1887 SIDEBOTHAM, EDWARD JOHN, M.B., Erlesdene, Bowdon, Cheshire.

†1848 SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., F.S.A., Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

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1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon, Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square.

†1842 SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1892 SIMS, FRANCIS MANLEY BOLDERO, 12, Hertford street, Mayfair.

1857 SIORDET, JAMES LEWIS, M.D., Villa Labrolles, Mentone, Alpes Maritimes, France.

1890 SMALE, MORTON, 22A, Cavendish square.

1879 SMITH, E. NOBLE, Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.

1891 SMITH, G. COCKBURN, M.D., 5, Inverness gardens, Kensington.

†1838 SMITH, HENRY SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1866 SMITH, HEYWOOD, M.A., M.D., 18, Harley street, Cavendish square.

1886 SMITH, HOWARD LYON.

1885 SMITH, JAMES GREIG, M.B., C.M., Lecturer on Surgery, Bristol Medical School; Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol.

1889 SMITH, ROBERT PERCY, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Southwark.

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1892 SMITH, SOLOMON CHARLES, M.D., 4, Portman Mansions, Baker street.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1872 SMITH, THOMAS GILBART, M.A., M.D., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.

*1874 SMITH, WILLIAM ROBERT, M.D., D.Sc., Barrister-at-Law, Professor of Forensic Medicine in, and Director of the Laboratories of State Medicine at, King's College, London; Medical Officer to the School Board for London; 74, Great Russell Street. *Trans.* 1.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1889 SPENCER, HERBERT R., M.D., B.S., Assistant Professor of Midwifery in University College; Assistant Obstetric Physician to University College Hospital; 10, Mansfield street, Portland place.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.

1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 6A, Bedford square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

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†1851 SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 53, Harley street, Cavendish square. *Trans.* 1.

1891 STEVENS, CECIL ROBERT, M.B., B.S., 11, Finborough road, Redcliffe gardens

1854 STEVENS, HENRY, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Falcon Lodge, Hampton, Middlesex.

1884 STEWART, EDWARD, M.D., Junior Carlton Club, Pall Mall.

†1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

*1879 STIRLING, EDWARD CHARLES, M.A., M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

†1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum; 4, Harley street, Cavendish square.

1871 STRONG, HENRY JOHN, M.D., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

†1863 STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1882-8.

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†1871 SUTHERLAND, HENRY, M.D., Physician to Newland's House and Otto House Private Asylums; 6, Richmond terrace, Whitehall.

1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 6.

1890 SYERS, HENRY WALTER, M.D., 3, Devonshire street, Portland place.

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.

*1890 SYMPSON, E. MANSEL, M.A., M.D., B.C., 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 6.

1875 TAY, WAREN, Senior Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Diseases of the Skin, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Hon. Secretary;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-92. *Sci. Com.* 1889—. *Referee*, 1887-8. *Trans.* 2.

1890 TAYLOR, SEYMOUR, M.D., Assistant Physician West London Hospital; 16, Seymour street, Portman square.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's. C. 1888-9.

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1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 11.

1890 THOMAS, WILLIAM ROBERT, M.D., Little Forest, Bath road, Bournemouth.

1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee*, 1876-7. *Trans.* 1.

†1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1891 THOMSON, JOHN ROBERTS, M.D., Monkchester, Bournemouth.

1892 THOMSON, ST. CLAIR, M.D., Florence.

1881 THOMSON, WILLIAM SINCLAIR, M.D., Hon. Surgeon, Kensington Dispensary; late Senior Consulting Surgeon to Peterborough Hospital, and Medical Officer of Health for Peterborough; 1, Palace court, Notting Hill gate.

1892 THORNE, WILLIAM BEZLY, M.D., 5, Gledhow gardens, South Kensington.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women and the New Hospital for Women; 22, Portman street, Portman square. C. 1891. *Lib. Com.* 1886-90. *Trans.* 5.

1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

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†1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 28, Weymouth street, Portland place.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1882 TOOTH, HOWARD HENRY, M.D., Physician to the Metropolitan Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen Square; 34, Harley street, Cavendish square.

*1871 TREND, THEOPHILUS W., M.D., Physician to Royal South Hants Infirmary; 1, Grosvenor square, Southampton.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Referee*, 1890—. *Sci. Com.* 1889—. *Trans.* 5.

*1881 TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1889 TURNBULL, GEORGE LINDSAY, M.B., 121, Ladbroke grove.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital, and to the North-Eastern Hospital for Children; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., The Lodge, Hemel Hempsted, Herts.

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1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Lecturer on Anatomy and Joint Lecturer on Practical Surgery at, St. George's Hospital; 49, Green street, Park lane.

1891 TWEED, REGINALD, M.D., 55, Upper Brook street, Grosvenor square.

1892 TWEEDY, JOHN, 100, Harley Street, Cavendish square.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Physician for the Diseases of Women, West London Hospital; 122, Harley street, Cavendish square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Pathologist and Curator of the Museum at the Middlesex Hospital; 13, Welbeck street, Cavendish square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., Assistant Surgeon, Charing Cross and Royal Westminster Ophthalmic Hospitals; 67, Grosvenor street, Grosvenor square.

1864 WAITE, CHARLES DERBY, M.B., Consulting Physician to the Westminster General Dispensary; 3, Old Burlington street.

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1884 WAKLEY, Thomas, jun., 5, Queen's Gate, South Kensington.

*1868 WALKER, ROBERT, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., 18, St. James's street.

1867 WALLIS, GEORGE, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. C. 1888-9. *Lib. Com.* 1882-5. *Trans.* 5.

*1883 WALTERS, JAMES HOPKINS, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.

1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 7, Hertford street, Mayfair.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1891 WARING, HOLBURT JACOB, M.B., B.S., B.Sc., St. Bartholomew's Hospital.

1877 WARNER, FRANCIS, M.D., Physician and Lecturer on Materia Medica and Therapeutics to the London Hospital; 5, Prince of Wales terrace, Kensington Palace. *Trans.* 1.

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1889 WASHBOURN, JOHN WYCHENFORD, M.D., Assistant Physician to, Physician in Charge of Electrical Department, Joint Lecturer on Physiology, and Demonstrator of Bacteriology at Guy's Hospital; Physician to the London Fever Hospital; Guy's Hospital.

1861 WATERS, A. T. HOUGHTON, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

†1861 WATSON, WILLIAM SPENCER, M.B., Surgeon to the Throat Department of the Great Northern Central Hospital; Surgeon to the Royal Eye Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., 30, Welbeck street, Cavendish square.

1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath.

1840 WEBB, WILLIAM WOODHAM, M.D., Neuilly-sur-Seine, France.

†1891 WEBER, FREDERIC PARKES, M.D., 10, Grosvenor street.

1857 WEBER, HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

†1844 WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

†1854 WELLS, SIR THOMAS SPENCER, Bart., Hon. M.D. Dublin, Leyden, Bologna, Charkoff; Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Foreign Associate, "Académie de Médecine," Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.

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†1842 WEST, CHARLES, M.D., Foreign Associate of the Academy of Medicine of Paris; Kenilworth, Eaton road, West Brighton. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Lib. Com.* 1892—. *Trans.* 4.

1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician City of London Hospital for Diseases of the Chest, Victoria Park; 34, Queen Anne street, Cavendish square. *Trans.* 1.

1882 WHARRY, CHARLES JOHN, M.D., 14, Ewell road, Surbiton, Surrey.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Senior Honorary Surgeon to the Kilburn Dispensary; "Madresfield," Acol road, Priory road, West Hampstead.

1875 WHIPHAM, THOMAS TILLYER, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892—.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888—. *Trans.* 3.

1890 WHITE-COOPER, G. O., M.B., 5, Cranley gardens, Brompton.

*1881 WHITEHEAD, WALTER, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; 499, Oxford road, Manchester. *Trans.* 1.

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*1885 WHITLA, WILLIAM, M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, Senior Surgeon to the Westminster General Dispensary, to the St. George's and St. James's Dispensary, and to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, The Hermitage, Clewer, Windsor. *Trans.* 1.

*1870 WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham, Kent.

*1883 WILKINSON, THOMAS MARSHALL, late Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 33, Avenue road, Grantham.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

*1883 WILLANS, WILLIAM BLUNDELL, Much Hadham, Herts.

1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.

†1865 WILLETT, ALFRED, *Trustee*, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

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1887 WILLETT, EDGAR WILLIAM, M.B., 25, Welbeck street, Cavendish square.

1888 WILLIAMS, CAMPBELL, 24, Welbeck street, W.

*1859 WILLIAMS, CHARLES, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., *Trustee for Debenture-holders*, Senior Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.

1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children; 25, Old Burlington street.

1872 WILLIAMS, JOHN, M.D., Physician Accoucheur to H.R.H. the Princess Beatrice; Professor of Midwifery, University College, London; Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Linden House, Bertie road, Leamington.

1890 WILLS, WILLIAM ALFRED, M.D., 23, Lower Seymour street, Portman square.

1887 WILSON, ARTHUR HERVEY, M.D., 504, Broadway, Boston, U.S.A.

1889 WILSON, JOHN HENRY PARKER, H.M.'s Military Prison, The Avenue, Brixton Hill.

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1889 WISE, A. TUCKER, M.D., Davos Platz, Switzerland.

*1850 WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn-Banbury.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

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1885 WOLFENDEN, RICHARD NORRIS, M.D., late Assistant Physician to the North-West London Hospital; Physician to the Hospital for Diseases of the Throat, Golden square; 35, Harley street, Cavendish square.

1887 WOOD, THOMAS OUTTERSON, M.D., 40, Margaret street, Cavendish square.

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., The Priory, Roehampton.

1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road, W.

1892 WOODHEAD, GERMAN SIMS, M.D., 1, Nightingale lane, Balham.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde park, Sydney, New South Wales.

1892 WRIGHT, ALMROTH EDWARD, M.D., Ch.B., Oakhurst, Netley, Hants.

1890 WYNTER, WALTER ESSEX, M.D., B.S., Assistant Physician, Middlesex Hospital; 30, Upper Berkeley street, Portman square.

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Regulations relative to the publication of the *Proceedings*.

The *Proceedings* are issued after each Meeting.

They are sent, postage free, to every Fellow of the Society, who in writing, expresses a wish to receive them.

They may be had by others at the Society's House, on payment (in advance) of an annual subscription of five shillings and eightpence, which may be sent either by post-office order or in postage stamps;—this will include the expense of conveyanc by post to any place within the Postal Union. For places beyond the Postal Union special arrangements must be made.

A notice of every paper will appear in the *Proceedings*. Authors will be at liberty, on sending their communications, to intimate to the Secretaries whether they wish them to appear in the *Proceedings* only, or in the *Proceedings* and *Transactions;* and in all cases they must furnish an Abstract of the communication.

The Abstracts of the papers read are sent to the Journals.

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ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

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ANNUAL GENERAL MEETING.

Tuesday, March 1st, 1892.

At 5 p.m.

Timothy Holmes, F.R.C.S., President, in the Chair.

Frederick Taylor, M.D.,
Warrington Haward, F.R.C.S., } Hon. Secs.

The Minutes of the last Meeting were read and confirmed.

The President nominated, as Scrutineers of the Ballot, Dr. Langley Browne and Mr. Bruce Clarke, and directed them to preside at the Ballot Table to receive the votes of Fellows until 6 *o'clock*.

The President then called upon Mr. Haward to read the

Report of the Council.

Since the last Annual Meeting the arrangements connected with the purchase of the Society's present

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house, and the alterations and additions made to it, have been completed; and the Building Committee which was appointed to carry out that business has presented its final Report.

This Report, which was presented to and adopted by the Council on January 27th, is as follows:

Report of Building Committee.

Since the last Annual Meeting of the Society the Building Committee have held nine meetings, and having now completed the work entrusted to them, they beg to present to the Council their final Report.

To the tenants enumerated in the last report the Huguenot Society and the Society of Medical Officers of Health have been added, and the Congress of Hygiene was accommodated during the period of its work for a fee of 100 guineas.

The annual rental of the Society now amounts to £2331.

The Royal Microscopical Society having now registered under the Literary and Scientific Society's Act, the vestry has admitted the claim of that Society to exemption from rates, which under the terms of their lease were payable by the Royal Medical and Chirurgical Society. This will save our Society about £20 a year.

The lease from the Corporation of London having been obtained, the Debenture Bonds were delivered to the holders in July last.

Certain serious defects in the drainage of Dering Yard having been discovered in August, it was found necessary to reconstruct the drain to Bond Street, the adjacent occupiers concerned, Messrs. Norman, Johnstone, and Co. and Mr. Ashdown paying a proportion of the expense. The Society's share in the cost of this work was £48 13*s*.

Some additional heating apparatus has been added

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in the Library, and some further precautions have been taken for the prevention of fire.

The contract for electric lighting, made with Messrs. Phipps and Dawson for £125 a year, having been found unremunerative by that firm, they exercised the right reserved to them in the agreement to terminate it at the end of the year, and a fresh contract has been made with them for two years at £180 a year.

The Building Committee think that it would be desirable to place on some part of the wall of the meeting-room a record of the date at which it was built.

The following statement shows the cost of acquiring and altering the new premises, and the manner in which the expense has been provided for:

Payments.			
Purchase . .	£23,000	0	0
Law Expenses . . .	537	9	2
General Building and Decoration .	12,646	12	2
Drainage	181	10	0
Electric Light . . .	1,690	0	0
Furniture	490	2	11
Fire Apparatus . . .	171	14	6
Removal	255	0	0
Architect	715	11	0
Sundries	106	6	0
	£39,794	5	9
Receipts.			
By Sale of Investments . .	£3,016	0	0
Loan raised on Debentures . .	36,000	0	0
Interest on deposit at Bankers .	42	5	4
Balance (provided from the General Fund of the Society) . .	736	0	5
Total .	39,794	5	9

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Having regard to the size of the new premises, the number of the Society's tenants, and the necessary business connected therewith, the Building Committee suggest to the Council the expediency of appointing a small Committee, to which questions of business unconnected with the Society's scientific work should be submitted, its decisions being reported to the next meeting of the Council.

In conclusion, the Committee wish again to acknowledge the very valuable services rendered to the Society by Mr. William Flockhart, the architect, and by Mr. Thomas Beaumont, the solicitor, by whose advice the Committee has been constantly guided; in addition to which the Committee desire to record the admirable and zealous manner in which they have been assisted in every detail of their work by the Resident Librarian, Mr. J. Y. W. MacAlister.

T. Holmes,

Chairman of the Building Committee.

The Building Committee is therefore now dissolved; and the Council have accepted its recommendation to appoint a small Committee (to be called the "House Committee"), to which questions of business unconnected with the Society's scientific work will be submitted, its decisions being reported to the Council at the next meeting following.

For this purpose the Council, in accordance with Bye-law, Chap. XIII, Sect. VII, have appointed the following Committee, viz.—The President and two Secretaries *ex officio* (the Senior Secretary to be Secretary of the Committee), Mr. T. Holmes, Mr. Willett, Mr. R. W. Parker, and Mr. Haward.

Particulars of the Income and Expenditure of the Society during the year 1891 are given in the Treasurer's Statement.

During the past year 53 new Fellows have been elected,

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of whom 38 are resident and 15 non-resident. Five Fellows have resigned, and 23 have died. The number of Fellows is now 422 resident, 381 non-resident; total, 803.

In accordance with suggestions made at the last Annual Meeting, the Council have submitted the Bye-laws to a careful revision, as a result of which the Council propose the following alterations to the Society:

In Chapter I, Section III, in Chapter II, Sections III and VI, and in Chapter V, Sections I, VI, and VII, the words "Seven miles of the General Post Office" shall, wherever they occur, be cancelled, and the words "Fifteen miles of the Society's house in Hanover Square" shall be substituted therefor.

In Chapter I, Section IV, the words "But the number of Resident Fellows who are general practitioners shall not exceed one third of the total number of Resident Fellows," at the end of that Section, shall be cancelled.

In Chapter V, Section I, line 8, the words "Forty-five," in line 11 the words "Thirty-nine," in line 13 the words "Thirty-three," in line 16 the words "Twenty-seven," and in line 19 the words "Twenty-one" shall be cancelled; and the words "Fifty," "Forty-five," "Thirty-nine," "Thirty-two," and "Twenty-four" shall respectively be substituted therefor.

In Chapter V, Section V shall be cancelled, and the following Section shall be substituted therefor, that is to say:

"V. Every person who shall, after the 1st day of March, 1892, be elected a Fellow of the Society, and does not live within fifteen miles of the Society's house in Hanover Square, shall pay to the Society the sum of Three Guineas as an Admission Fee, and shall, after the first year from his admission (the said Admission Fee being deemed to include his first year's annual contribution), contribute the sum of One Guinea annually, and shall be entitled to receive the

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Proceedings of the Society, and to consult books and periodicals in the Reading Room, and to borrow one volume at a time from the Library. Any Non-resident Fellow wishing to enjoy the full privileges of a Resident Fellow shall be at liberty to do so on paying the annual contribution of Three Guineas."

In Chapter V, Section VII, the words "and freed from the payment of his annual contribution" shall be cancelled, and the words "and be only liable to pay the annual contribution of a Non-resident Fellow" shall be substituted therefor.

In Chapter XVI, Section II, line 5, the word "any" shall be cancelled, and the word "every" substituted therefor.

In the Appendix, form No. 2, the word "Six" in the first paragraph shall be cancelled, and the word "Three" substituted therefor; and the third paragraph shall be cancelled and the following paragraph substituted therefor, that is to say: "I am further to inform you that you will be at liberty to avail yourself of all the privileges of a Resident Fellow by increasing your annual contribution of One Guinea to Three Guineas instead of paying the Eight Guineas referred to."

In Chapter XVII, Section I, line 6, the words "half-past seven" shall be cancelled, and the words "twenty-five minutes past eight" substituted therefor.

In Chapter XXI, Section II shall be cancelled, and the following section shall be substituted therefor, that is to say:

"II. The Charter, the Common Seal, and the Deeds of the Society shall be kept in safe custody in such manner as the Council shall from time to time direct."

The Hon. Librarians report as follows:

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Report of Honorary Librarians.

"The past year has been an uneventful one in the history of the Library. The Committee, released from the necessity of economising, which crippled their purchasing powers during the heavy expenditure on building, have added to the stock of books nearly 200 volumes, exclusive of the many volumes of periodicals and serials which have been kept up to date, in addition to which, by gifts, they have received nearly 300 volumes and pamphlets. These include a most valuable gift received from the Executors of the late Dr. Wilson Fox—through Dr. Sidney Coupland—consisting of a large collection of valuable pamphlets on the lungs. The collection is bound in ninety volumes, and is accompanied by a carefully written catalogue which greatly enhances the value of the gift.

"A recently arranged change in the tenancy of the Royal Historical Society releases the shelving on the south wall of the north room, and thus places at our disposal the whole of the shelving in that room. This will enable the Librarian to place more conveniently a large number of volumes that have hitherto been kept upstairs.

"The binding of periodicals and new books, and the rebinding of many old ones, have been carefully attended to.

SAMUEL J. GEE,
J. W. HULKE,
Hon. Librarians.

The Committees appointed to investigate the Climatology and Balneology of Great Britain and Ireland, and that for considering the Treatment of Suspended Animation in the Drowned, are still at work.

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In conclusion, the Council desire to express their appreciation of the ready and valuable assistance which they have always received from the Resident Librarian, Mr. J. Y. W. MacAlister.

The PRESIDENT then called upon the Treasurers to submit their accounts.

Mr. BOSTOCK, in presenting the statement of moneys received and expended on the General Account, offered the following explanation of some of the items:

"The audited account shows, as required by the Bye-laws, the moneys actually received and expended in the General Account of the Society during the year ending 31st December, 1891, but it contains nothing belonging to the Building Account. The latter cannot be conveniently audited until the Building Committee are in a position to close it.

"When the Building Account is presented it will supplement and complete the statements rendered from 1889 to the end of 1891, and taken with these will show the entire receipts and expenditure during those three years. For example, in the account now presented, the total sum paid during 1891 for interest on debentures appears to be only £653 18*s*., but this merely represents the interest due for the half-year ending July 1st, 1891.

"The previous half-year's interest (due January 1st, 1891), amounting to £607 15*s*. 4*d*., was paid out of the Building Account in order approximately to restore to the General Account of the Society the first payment of interest, £668 17*s*. 4*d*. (1st January, 1890), properly chargeable to the Building Account, but temporarily advanced out of the general fund. In addition to this

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amount advanced for interest it will be observed that the Report of the Building Committee states that a sum of £736 0*s.* 5*d.* is chargeable to the General Account for building and other purposes.

"As the Receipts and Payments Account has been already circulated we propose that it be taken as read, and we are pleased to say that one of our auditors (Mr. Mundy) is present, who will willingly answer any questions as to accounts."

Dr. HARE added some further explanation as to the accounts.

Mr. HARRISON CRIPPS, Mr. BRUCE CLARKE, Mr. CHRISTOPHER HEATH, and Mr. WILLETT criticised the accounts and asked several questions, which were replied to by Dr. HARE and the professional auditor (Mr. MUNDY).

Resolved on the motion of the PRESIDENT, seconded by Dr. GEORGE HARLEY—"That the Report of the President and Council, together with the Treasurer's audited Statement of Receipts and Payments, be adopted and published in the next volume of *Transactions*."

Resolved on the motion of Mr. GEORGE POLLOCK, seconded by Mr. J. H. MORGAN—"That the very cordial thanks of the Royal Medical and Chirurgical Society be given to the members of the Building Committee, for the unremitting care which they have devoted to the interests of the Society during the last three years."

The PRESIDENT, as Chairman of the Building Committee, replied.

Resolved on the motion of the PRESIDENT, seconded by Dr. GEE—"That the alterations of the Bye-Laws made by the Council, of which notice has been given, and which are fully set forth in the circular summoning this meeting, be and are hereby confirmed."

The PRESIDENT then delivered his ANNUAL ADDRESS.

Resolved on the motion of Sir EDWARD SIEVEKING,

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seconded by Dr. DOUGLAS POWELL—"That the very hearty thanks of the Society be given to the retiring President, Mr. Timothy Holmes, for his zealous discharge of the duties of his office, for the Annual Address now delivered, and also for his great and valuable services as Chairman of the Building Committee, throughout the whole period of its important labours."

The PRESIDENT replied.

Resolved on the motion of PROFESSOR ALLBUTT, seconded by Dr. BLANDFORD—"That the best thanks of the Society be given to the retiring Vice-Presidents, Dr. Langdon Down and Mr. Alfred Willett, for their services to the Society during the past year."

Resolved on the motion of Mr. WILLETT, seconded by Dr. NORMAN MOORE—"That the cordial thanks of the Society be given to the retiring Honorary Secretary, Mr. Warrington Haward, not only for his assiduous discharge of the duties of that office, but for his exceptional and most valuable services to the Society during a most critical period of its history as Honorary Secretary of the Building Committee, from its formation to its dissolution."

Mr. HAWARD replied.

Resolved on the motion of Dr. CHURCH, seconded by Mr. BRUCE CLARKE—"That the best thanks of the Society be given to the retiring members of Council, Dr. Cheadle, Dr. Gowers, Dr. Vivian Poore, Dr. John Williams, Mr. Harrison Cripps, Mr. H. W. Kiallmark, Mr. Page, and Mr. Knowsley Thornton, for their valuable services to the Society during their respective terms of office."

The Scrutineers reported that the following gentlemen had been duly elected as officers and members of Council for the ensuing year:

President.—Sir Andrew Clark, Bart., M.D., LL.D., F.R.S.

Vice-Presidents.—Walter Selby Church, M.D.; George Harley, M.D., F.R.S.; F. Howard Marsh; Henry Power.

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Honorary Treasurers.—Charles John Hare, M.D.; John Ashton Bostock, C.B.

Honorary Secretaries.—Frederick Taylor, M.D.; Rickman J. Godlee, M.B., B.S.

Honorary Librarians.—Samuel Jones Gee, M.D.; John Whitaker Hulke, F.R.S.

Members of Council.—Thomas Barlow, M.D.; John Mitchell Bruce, M.D.; Alfred Lewis Galabin, M.A., M.D.; Norman Moore, M.D.; Thomas Tillyer Whipham, M.B.; J. Neville C. Davies-Colley, M.C.; Alfred Pearce Gould, M.S.; Sir Joseph Lister, Bart., D.C.L., LL.D., F.R.S.; Charles N. Macnamara; William Henry Brace, M.D.

The President then invited Sir Andrew Clark, the President elect, to assume the Presidential Chair, and transferred to him the badge of office and the silver master-key of the Society's House.

Sir Andrew Clark, on taking the Chair, briefly addressed the Meeting, and thanked the Fellows for the honour bestowed upon him.

This terminated the proceedings.

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RECEIPTS.	£ s. d.	£ s. d.
To Balance on 1st January, 1891 :		
Cash in hand	27 15 9	
„ at Bankers	424 18 4	
		452 14 1
„ *Subscriptions, Fees, &c.* :		
392 Annual Subscriptions at £3 3*s*.	1234 16 0	
55 Entrance Fees at £6 6*s*.	346 10 0	
2 Non-resident Subscriptions at £1 1*s*.	2 2 0	
3 Composition Fees for *Transactions* at £8 8*s*.	25 4 0	
3 „ (Life)	77 14 0	
Fines	3 0	
		1686 9 0
„ *Transactions* and *Proceedings* :		
Sold by Messrs. Longmans (*Transactions*)	41 13 5	
„ Librarian (*Proceedings*)	1 7 0	
„ „ (Catalogues)	1 2 6	
		44 2 11
„ Rents received		2646 6 3
„ *Interest* :		
On Permanent Endowment Fund	12 14 6	
From Prudential Insurance Co.	33 18 8	
		46 13 2
„ Miscellaneous Receipts		2 17 0
		£4879 2 5

CHARLES J. HARE, M.D.,
J. A. BOSTOCK, } *Treasurers.*

[1] The other half-year's interest (1st January, 1891) was paid out of the

PERMANENT

(TRUSTEES: *Sir Andrew Clark, M.D., Walter*

New South Wales "Four Per Cents."

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PAYMENTS.	£ s. d.	£ s. d.
By Rent, Rates, Taxes, &c.		229 11 5
„ Rates paid for Royal Microscopical Society		29 6 3
„ Lighting, Cleaning, and Heating		298 19 9
„ Fire Buckets		7 6 0
„ New Clocks for Meeting Rooms and Libraries		43 19 0
„ Furniture, Repairs, &c.		37 2 10
„ Framing of Portraits		19 8 6
„ Meeting Expenses		20 5 4
„ *Transactions* and *Proceedings*		321 8 7
„ Stationery and Sundry Printing		122 2 0
„ Stamps, &c.		29 16 6
„ Salaries and Wages		737 18 6
„ Library :—Books, and Binding		439 18 7
„ *Extraordinary Charges :*		
Conversazione	149 3 9	
Gratuities (Richard Coldrey, £25 ; Alfred Tapson, £5) ..	30 0 0	
"Spas" Committee	16 9 7	
		195 13 4
„ Transferred to Building Account		200 0 0
„ Interest on Debentures (for one half-year : 1st July, 1891)[1] ..		653 18 0
„ Annuity		34 16 0
„ Cheques, &c.		1 9 3
„ Miscellaneous Payments		5 12 8
„ *Balance :*		
Cash in hand	172 19 5	
At Bank—Current Account .. £1256 8 6		
„ Coupon „ .. 21 2 0		
	1277 10 6	
		1450 9 11
		£4879 2 5

Audited and approved :
H. WOODBURN KIRBY & Co., Chartered Accountants, } *Auditors.*
19, Birchin Lane, E.C.
22nd February, 1892.

Building Fund in order to restore in part the advances from the General Fund.

ENDOWMENT FUND.

Butler Cheadle, M.D., and Christopher Heath).

.. .. .. £326 7 3

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ADDRESS

OF

TIMOTHY HOLMES, F.R.C.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1st, 1892.

Gentlemen,—It is not to be wondered at that a year so peculiarly fatal as this has been, and especially to persons past the prime of life, should have caused more gaps in our ranks than usual. The number of deaths in this period was twenty-five, as contrasted with nineteen in the previous year, and thirteen in the year before last. None of these were Foreign or Honorary Fellows; thirteen were resident[1] and twelve non-resident.

I shall speak of them in the order of their decease:—The first on our list is *Mr. Richard Middlemore*, of Birmingham, who died on the first day of our year, March 1st, 1891, aged eighty-six. He had a very high reputation in Ophthalmic Surgery—a branch of practice which he followed with enthusiasm and with conspicuous success, along with the hard labour of a large general practice, till he felt the necessity of retiring at the age of seventy-five. But even when he could no longer practice his profession, he never

[1] I have reckoned Sir Prescott Hewett as a resident Fellow, though his residence was in the country; for he still appeared amongst his London friends, and all his work at the Society was done as a resident Fellow.

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lost his interest in it, and he showed that interest by the substantial benefits conferred on the profession during his life; viz. his prize for ophthalmology, awarded triennially by the British Medical Association; a grant of £1000 to endow a course of lectures in ophthalmology at the Birmingham and Midland Eye Hospital, and another of £2000 to the Birmingham Asylum for the Blind.

Mr. Middlemore was one of the few surviving links between our day and the time of Abernethy, whose dresser he had been, and by whom he was recommended to the notice of Mr. Hodgson. The venerable Sir Richard Owen was his friend at St. Bartholomew's Hospital, and continued so during his long life.

One who knew him writes:

"Throughout the whole of his life his earnest love for and devotion to his profession, and his generous unostentatious sympathy, endeared him to those associated with him. Conscientious in his relations with his professional brethren, modest, never seeking notoriety, simple, kind, generous, sensitive to a fault, always maintaining a high standard of professional life, he has furnished his survivors an example which we shall do well to follow. Of him and his teaching it may well be said, 'Memoria bene redditæ vitæ sempiterna.'"

Dr. Heale was long in practice at Winchester, and after his retirement, lived at St. Leonard's where he died at the age of eighty. Dr. Heale was a pupil at the old school at Webb Street, and afterwards at St. George's Hospital. He was an ingenious mechanician, and when I knew him used to spend much time in the dissecting-room at St. George's Hospital, occupying himself with physiological experiments, and especially with the construction of a most elaborate machine for fine and coarse injection, which, however, was intended as a monument of its author's knowledge and ingenuity, rather than for practical use. He was a kind-hearted, generous man, and obtained the friendship of those to whom he was known; but he had long ceased to have any active connection with the medical profession.

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Dr. Crompton was a well-known practitioner at Manchester, and had a family connection with the manufacturing districts and with ophthalmic surgery, for his uncle was the inventor of Crompton's "mule jenny," which followed close on the invention of Hargreaves' spinning jenny; and another uncle was an ophthalmic surgeon, celebrated in his day at Manchester. It was accordingly in the latter city that Dr. Crompton started in practice, and he soon became surgeon to Henshaw's Blind Asylum. His only recorded work was a treatise on 'The Causes of Blindness.' He acquired a good standing and much popularity at Manchester, from whence he retired ten years before his death, and spent the remainder of his life at Cranleigh, in Surrey. He is spoken of as a man of elegant tastes and varied accomplishments.

Mr. Robert C. N. Davies died on May 9th, aged sixty-one. He settled in practice at Winchester in 1850, where he was coroner and mayor, and moved thence, two years afterwards, to Rye in Sussex, where he was for ten years medical officer to a large union district, and occupied a distinguished position as justice of the peace and alderman. His practice was successful and his reputation good; but for the last ten years he had been in failing health from repeated attacks of paralysis, the last of which proved rapidly fatal.

Dr. Steavenson, who died on June 1st, at the early age of forty-one, was a good instance of the usefulness and success which may attend a life, even when it is carried on under the pressure of constant ill health. As a well-informed friend of his has said in the 'British Medical Journal':—"From childhood the victim of spasmodic asthma, and the subject of a certain degree of emphysema as a result, Dr. Steavenson never enjoyed robust health, but he never, if he could possibly help it, allowed the state of his health to interfere with the punctual and efficient discharge of his professional duties, and many a time he was to be seen at the hospital when his asthma was quite bad enough to have made any ordinary person shirk work.

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"His indomitable courage in this respect gained for him the admiration of all who were brought into contact with him, whilst his gentleness of manner and ready sympathy secured the lasting affection of those who were privileged to know him intimately. In all questions of medical politics he took the deepest interest, and was always prepared to state and uphold his views even when in a distinct minority."

As I happened to sympathise with some of these views of the "minority," I saw a good deal of Dr. Steavenson in connection with them, and also at the Charity Organisation Society, and other not strictly professional institutions, as well as at the Children's and Alexandra Hospitals, where his medical acumen and his practical sagacity made his services most valuable. He was a most judicious counsellor, a man of the highest feeling and honour, and one who rose higher in the esteem of his friends the longer and the more intimately he was known.

It was in the application of electricity to medicine that Dr. Steavenson had made his special reputation, and here he had become a recognised authority, having obtained the high position of physician-electrician to St. Bartholomew's, the hospital at which he had received his medical education, and where he had served as house surgeon, before he went to Cambridge. Dr. Steavenson was well known to all medical graduates of Cambridge as one of the founders and original secretaries of the "Cambridge Medical Graduates' Club," a body which owes no little of its prosperity to his wise and efficient guidance.

Dr. Steavenson had connected his name with one of those great in the annals of surgery by his marriage with the granddaughter of the famous Benjamin Travers, a union too early dissolved by his death. This followed on an attack of influenza and bronchitis, which his weak frame was but too little fitted to resist.

He had not been a Fellow of our Society for any great length of time, and had not borne office, nor contributed to our 'Transactions.' Had his life been spared, there was no

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one from whom we might have more confidently looked for good work in both these departments.

Dr. Henry G. Sutton, Physician to the London Hospital, was one of the many victims to the disease which has lately prevailed so extensively, dying of pneumonia after influenza, at the age of fifty-five.

He did not join the Society till after he had given the one contribution to our 'Transactions,' which bears his name only, in the shape of a paper on "Fibroid Degeneration of the Lungs," communicated by Dr. Barlow in 1865.

He was a student of King's College and a graduate of the University of London, but connected himself with the London Hospital as assistant physician in 1867, and as physician in 1876. No one was more appreciated and more beloved by the students, for he was devoted to clinical work and to clinical exposition. One who knew him well as a colleague, speaks thus of his hospital work and of his personal character :—"Dr. Sutton always struck me as a man with a large, an original, and active imagination; he was wonderfully *magnetic,* attracting men to him and to his work, inspiriting them by his kindly interest and suggestions, not less by his marvellous prognostic powers. He would examine a case, dictate notes on it, diagnose, prognose, pathologise, and in fatal cases, read out these notes in the post-mortem room and compare his forecast with what was actually found at the autopsy. For many years his Sunday mornings were spent in the hospital, and he was accompanied in his rounds by a class of resident officers and senior students, who almost worshipped him.

"His great misfortune was to be deaf, which put him often outside ordinary conversation, and made him appear as if indifferent, even daft at times. He was (perhaps on this account) thrown back on himself, and hence developed a contemplative turn of mind. He was fond of the country, of fishing, of flowers, of birds, of poetry. Very gentle, very considerate; with a powerful memory, he made his teaching very attractive, and contrasted cases one with another."

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A distinguished physician permits me to incorporate with this very scanty notice of a man of rare merit, the following appreciation of Dr. Sutton's published work :—

"Dr. Sutton's writings, like his employments, were both pathological and clinical; but pathology was his chosen subject and the chief work of his life. In clinical medicine he wrote enough to have established a considerable reputation, if he had had nothing else to rest it upon.

"Dr. Sutton was the author of many papers, one in the 'Transactions' of this Society on "Fibroid Degeneration of the Lungs," and several elsewhere, relating chiefly to pathological matters; but probably he will be longest remembered in connection with those in which the authorship was shared with Sir W. Gull.

"The first of these, on 'The Natural History of Rheumatic Fever,' presented the conclusion that this disease was as favourably treated by mint-water as by anything else, that as to alkalies, it made little difference whether these were given or withheld, and that the part of the physician with regard to acute rheumatism was that of a spectator. Some who habitually use alkalies in this disease may hesitate to accept the conclusion, and may even think that it was scarcely justifiable to leave rheumatic fever to its own course, in order to find out what that course was; but this is a matter of opinion only, and depends on the degree of belief which each entertains as to the efficacy of drugs under the circumstances. Of this belief Gull and Sutton had little or none. The other paper in our 'Transactions,' under the same joint authorship was on "Chronic Bright's Disease with the Contracted Kidney," and involved descriptions of the changes in connection with the disease which suggested as a more appropriate name for it, that of "Arterio-Capillary Fibrosis." This paper, which sought to show that the vascular changes occurring with the granular kidney were rather fibrotic than muscular, rather coincident with the renal disease than consequent upon it, was followed up by one in the 'Pathological Transactions,' which bore especially upon the spinal cord and reiterated the conclu-

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sions formerly arrived at. These papers gave rise, as might have been expected, to animated discussions which on the present occasion cannot properly be reopened or pronounced upon; but whatever may be thought now, or whatever may be the verdict of the future, as to the conclusions to which Dr. Sutton, together with Sir W. Gull, was led, there can be no difference of opinion as to the skilful labour and truthful purpose with which Dr. Sutton accumulated the large mass of pathological observations on which the papers were founded. It is no disparagement to his coadjutor to infer that the groundwork of observation was chiefly supplied by Dr. Sutton.

"But the papers which Dr. Sutton wrote or took part in, whatever evidence they give of conscientious research, convey a very incomplete idea of his mental qualities. He was no dull collector of facts or mere laborious drudge. All who have read his admirable 'Lectures on Medical Pathology,' which may be regarded as his most mature and comprehensive work, and above all, those who were acquainted with him personally, know how wide was his range, and how philosophical his manner of thought, how much he possessed of imagination, and even of poetic sentiment."

Dr. Felix W. Lyon, of Edinburgh, died on June 5th, at the age of eighty-seven. I have not been able to ascertain any particulars of his career, nor does he seem to have left any medical or other works behind him. He studied at the University of Edinburgh, and at Guy's Hospital. At the latter institution he was a contemporary of Dr. Wilks and Mr. Bryant, and was, therefore, a man of forty or forty-five years of age when a student. He seemed to be a man of means, and not to be under the necessity of living by his profession. He is believed to have filled some private appointment in the south of England before returning to his native country.

Sir Prescott Hewett, Bart., who died on June 19th, aged about seventy-nine, was one of the most distinguished of our Fellows, one of the most distinguished surgeons, indeed, of our time. Born in 1812, the son of a Yorkshire country

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gentleman, he was educated in France, and to the last spoke French with a fluency and correctness of accent which are only too rare amongst Englishmen. I have been informed that in Paris he was at first educated for the profession of painting, and it was from this artistic education, probably, that he acquired that talent for water-colour drawing which was his own unfailing resource in his vacations, and the delight of his friends at all times. He soon, however, abandoned the idea of making art his profession, if he had ever seriously entertained it; and "had the good luck to become intimate with the son of a successful Parisian surgeon, by whom he was first inspired with a love for that noble profession in which he was destined to achieve the highest distinction. After being thoroughly grounded in the principles and practice of French surgery—which to the end of his days he regarded as the neatest and most resourceful in Europe—Sir Prescott attached himself to St. George's Hospital in London, and became a member of the Royal College of Surgeons in 1836. For many years he laboured hard, but unostentatiously, in the practice of his profession, to which he devoted himself with a singleness of purpose and an untiring energy which seldom fail to lead to success in any walk of life." So speaks a well-informed writer in one of the daily papers. And, indeed, Hewett had to "labour hard," for success did not come to him except as the result of patient and untiring devotion to the actual work of his profession. Of all the petty artifices by which success is sometimes reached by the undeserving, he was not so much disdainful as incapable. Still, though his progress might be slow, it was sure, and he gradually became known, first to professional circles as one of the most profound anatomists, and one of the best lecturers in London (a matter in which his artistic skill was of the greatest service to him) then as a most zealous pathologist, a follower of Brodie in his constant reference of the phenomena of disease in the living to the records of the deadhouse, then as an organiser of rare energy and power, and ultimately as one of the most

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accomplished of living surgeons, and one of the most admirable of operators. Having had in my early days of surgery unusual facilities for seeing the operations of all the leading surgeons of London, I may say that, though some might perhaps excel him in what may be called "display," no one appeared to me to perform his operations with more perfection of detail; there was no one in whose hands a patient seemed more safe. And, though he was by no means rash, or fond of operating for the operation's sake, yet he shrank from no necessary proceeding, however formidable. Nor was he less skilful in the, perhaps higher, department of surgery—diagnosis. I, who watched Hewett's practice from a very early period of his appointment as assistant surgeon to St. George's till the close of his career, had plenty of opportunity of seeing how rare were his mistakes (though, like all the sons of men, he was not actually free from them), how careful and methodical he was in following up every point of a case, and how often he came to the right conclusion in difficult cases which had puzzled or misled others. Many striking instances of this occur to me, but this is hardly the place to give them. As a teacher he was equally admirable, "and he was one of the fittest men in the world to instruct students; for he had all the lucidity of exposition which is required to convey the knowledge of subjects teeming with difficulties of detail, his ready pencil would illustrate the most complicated anatomical descriptions, and his stores of experience could furnish cases in point in all discussions. But, far above all this, he was a man of that high character, that lofty sense of honour, which readily finds a response in the bosoms of the young, and gives to its possessor an influence over them which no merely technical excellence can ever bestow."

His connection with the hospital school commenced very early in his career; for at the time of his return to England from Paris the school was somewhat disorganised, and the condition of its pathological department hardly what would have been expected in the hospital of Brodie. This

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department was put under Hewett's management as Curator, and he rapidly not only reduced it to order, but introduced there a system of keeping the records of cases and post-mortem examinations which is followed there to this day, and has been imitated at many similar institutions.

"As assistant surgeon he worked with the same vigour which he brought into all that he undertook, and his talent for organisation was further displayed in the admirable arrangements which he devised for the hospital register—a department in which he was aided by the late Dr. Barclay—and which he further developed in after years by introducing tables for the preservation of the continuous experience of the hospital in amputations, operations for strangulated hernia, and compound fractures. The first set of tables especially have been found of the greatest value both in reference to hospital hygiene and surgical practice. Two papers dealing with 500 cases were published by me in the 'St. George's Hospital Reports;' and a third by Mr. Dent and Mr. Bull, carrying on the series to 900 cases, in the 73rd volume of the 'Med-Chir. Trans.,' resumes the whole subject."

He had many years to wait for promotion to the office of full Surgeon to the hospital to which he succeeded, on the retirement of Mr. Cæsar Hawkins in 1861. By that time he was rising rapidly into great practice. He became President of the College of Surgeons in 1876, and was made a Baronet in 1882. On Sir W. Fergusson's death he was promoted to the rank of Sergeant-Surgeon Extraordinary, and to that of Sergeant-Surgeon on Mr. Cæsar Hawkins' death. Long before this time, however, his surgical position had acquired for him the personal confidence of the Queen and the Prince of Wales, and he was constantly consulted in any surgical emergency occurring to members of the Royal Family. Shortly after receiving his title he retired from London, and "though he was still at the service of his old patients on emergencies and paid periodical visits to London for professional purposes, he gave up most of his time to country pursuits, which he

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loved, and to painting, which he loved still more. His life here was an eminently happy one. Few men of his age could match him in activity and vigour, nor would any one who met him then for the first time have suspected that he had already passed the limit of three-score years and ten.

"He delighted in the society of his family and of his old friends, and had everything about him that should attend an honoured and beloved old age. Shortly before his death, however, troubles came thick upon him. The illness of his only son was a grievous trial to him, and soon afterwards it became evident to those about him that his own end was approaching. He bore his sorrows, however, with the patience and the unfaltering courage which he had displayed in all the former trials of his life, and endured uncomplainingly the terrible agonies of a disease which he must have known to be mortal, though for the comfort of his family he professed to make light of them, and to refer them to a temporary cause."

His son did not long survive him, and, as he was childless, the title becomes extinct.

Sir P. Hewett's contributions to our 'Transactions' were numerous, but they were more remarkable for their value than their number. Some of them have formed the commonplaces of surgical writers on the subjects of which he there treated ever since their publication, such as his first paper in vol. xxvii, which he communicated to the Society in the first year of his fellowship, and while still merely curator of St. George's Hospital Museum. That paper dealt with the very important subject of "omental sacs" in strangulated hernia, and the facts on which it rests, together with the operative method prescribed, have guided many an operator through an operation which might otherwise have ended in discredit to him and disaster to his patient. To this paper Mr. Hewett added a tabular statement of all the operations for hernia performed at the hospital during the previous two years, the commencement of the tables first suggested by him, and begun by myself when Surgical Registrar at St. George's, in which

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the whole experience of the hospital for now nearly forty years in this important branch of surgery is contained. Such again was the famous paper on membranous extravasations in the arachnoid cavity, published in the following year (vol. xxviii). Both these papers illustrate the valuable contributions which may be made to practical surgery by a man who has hardly commenced practice on his own account. Of still greater value, in my judgment, is the comprehensive paper in vol. xxxvi, in which he summarised the cases of injury of the head which had been examined during ten years at St. George's Hospital, and in which, among many other points of importance, he so clearly proved that the usual source of the watery discharge from the ear in fractures of the base is the cerebro-spinal fluid, and so sagaciously anticipated that this would prove not to be its only source: and in accordance with his conjecture it has now been proved that in come rare cases the fluid flows from the ventricles. Two others of Mr. Hewett's papers (in vol. xxix, and vol. xxxiv, p. 161) refer to aneurysms, a subject in which he had always a vivid interest. Another (vol. xxxiv, p. 43) is an interesting account of a fatal attempt to extirpate a tumour of the upper jaw, related with a view to its bearing on the diagnosis of tumours in this region. The remaining paper (vol. xxx) relates to the somewhat remote subject of cancer of the walls or lining membrane of the heart.

Such is a very brief summary of Hewett's contributions to our 'Transactions.' He was active in this Society in other ways also—a constant attendant at one time, and a frequent speaker at its debates; a member of the Council in 1859, and Vice-President in 1866, 7; a member of the Library Committee in 1846, 7; of the Chloroform Committee in 1863; and a referee of papers for a great number of years, in fact, till he left London. And it is no secret that the fact that his name does not appear in the list of our Presidents, was due to his own hesitation to undertake an office for which at the time he had hardly sufficient leisure, and not to any want of appreciation on

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the part of the Council of that day of the honour and advantage which the Society would have gained from his presidency.

Sir Prescott Hewett was not a voluminous writer, "for he shrank with horror from anything in the nature of self-advertisment, and his taste was fastidious, especially in his own case. His intimate knowledge of the French language and his old connection with Paris naturally brought him a thorough acquaintance with French surgical literature; and like so many of the great French surgeons, he was particularly drawn to the study of injuries and surgical affections of the head. His lectures, as Professor at the College of Surgeons on this subject, were unfortunately never published in their entirety, though portions of them formed the base of some valuable articles on encephalocele and kindred affections in the 'St. George's Hospital Reports.' His article on "Injuries of the Head" in the 'System of Surgery' edited by me is also one of the standard authorities on that subject. Aneurysm, again, was a very favourite subject with him, and he took the keenest interest in the treatment by compression when this was comparatively novel. In pathology the work he did was most valuable and extensive. He was an original member of the Pathological Society, and deeply interested in its success. Amongst the numerous distinguished men who have presided over that Society, few had done more service to it than Hewett, and to its younger sister—the Clinical—he also gave his great services as President, and inaugurated that remarkable discussion on "Pyæmia in Private Practice" which did so much to refute the exaggerated representations then in vogue as to the insalubrity of our hospitals.

This is no meagre catalogue of good and solid work. Still we must confess that his extensive experience and his admirable knowledge, both of literature and practice might have given us more; and now that his living voice is silent, we can only mourn that he has not left us still larger treasures of his ripe wisdom.

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Of his private character it is hardly necessary to speak. I was a pupil and an intimate friend of his, bound to him by all the kindly recollections which surround an old teacher, and by the memory of many acts of generous kindness, and many occasions in which he gave his time and his matured wisdom to help one who was then trying to follow in the upward path which he himself had trod so bravely. It would be impossible, therefore, for me to speak critically or dispassionately of the character of my old friend and master; but fortunately it is not necessary. Few men were better known in the professional or social world than Hewett, and none more widely respected and liked. The reason is that he was emphatically a gentleman —a man who would not merely scorn a base action, but with whom anything base would be inconceivable. He was a great surgeon, and knew his art as thoroughly as he knew his anatomy; but he was much more than that. He was one of the most ardent and generous of friends, one of the truest and most reliable of colleagues, a man of whom his school, his hospital, and his profession were justly proud.

The youngest, and one of the most accomplished, of our Fellows was *Dr. Chas. E. Sheppard,* who died on June 30th, at the early age of thirty-five.

Dr. Sheppard had only just joined this Society, and, therefore, had had no time to enrich our 'Transactions' with the products of a mind of rare promise. But he had just time to show that he had merits which must have brought him into a distinguished position, if fortune had favoured him. He earned the Solly Medal at St. Thomas's Hospital by an essay which remains his only published work, beyond some communications to the journals on the subject of anæsthetics, a department of practice in which he had always been much interested, and which lately he had began to follow with great and increasing success, till a sad accident which occurred in his practice wrought on his too sensitive mind, so as to deprive him of reason, and bring on a fatal calamity. But it was not in medicine

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only that Dr. Sheppard was distinguished. A friend of his, writing in the 'British Medical Journal,' gives the following attractive description of the varied talents of one too early lost to the Society and the profession:

"He was an accomplished draughtsman, as the two plates published with his essay testify. But brilliant as Charles Sheppard was in his profession, he had the most surprising all-round knowledge. He seemed to know everything. It was no ordinary superficial knowledge, but one extending to the most abstruse and technical details. It would have occasioned no surprise to be told that he could read Chinese or decipher the Egyptian hieroglyphics. His mind was a complete encyclopædia.

"His knowledge of literature was wide. He was a great book collector, and probably knew as much about rare books as many professed bibliophiles. During the time when he had given up professional work he made a complete glossary to Burns, and for this purpose he studied very thoroughly the early Scotch poetry. His wonderful versatility was, however, nowhere more apparent than in music. Few professional pianists could excel him in brilliancy of execution, in lightness of touch, or in depth of feeling. Of late he frequently played at private concerts, but he was always at his best when he had only a friend or two to listen to him. His impromptus were then sometimes marvellous. His fantasias on popular airs will always live in the memory of those who heard them. How many instruments he could play probably no one but himself knew.

"He was not satisfied with being able to play on instruments, but he learnt the principles of their construction. He built an organ at one time entirely with his own hands. No evening passed for him without music, and generally an hour after dinner was devoted to it. Chopin was probably his favourite composer, and his interpretation of his works was wonderfully sympathetic and original. He had a most thorough knowledge of harmony, counterpoint, and composition, and it was surprising how quick he was to

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detect the smallest error in printed scores. He often used to say that his musical talent was not to him an unmixed good. It made the discords of the world more jarring and more obtrusive, and increased his natural sensibility."

Dr. Mervyn Crawford died on June 29th, at the age of eighty-four. He had been long retired from London practice, but at one time occupied a distinguished position at the Middlesex Hospital, where he became Assistant Physician in 1841, full Physician in 1843, and Lecturer on Medicine in 1845. He was also Councillor and Censor at the College of Physicians. He quitted London in 1855, and from that year to 1876 is said in our lists of Fellows to have resided at Wiesbaden; but I have failed to find any record of his practice there, and I believe that he also carried on practice during a portion of the year at Paris. Finally he returned to England, and resided at Brighton from the year 1880. He served on the Council of this Society in 1853–4. I regret that I can give no further particulars of Dr. Crawford's career.

A remarkable man of vigorous intellect, and one whose career had a substantial influence on the progress of medicine, passed away last year in the person of *Dr. James Henry Bennet*.

He had long been obliged by threatenings of lung disease to reside abroad, and had for some years before his death retired from practice; but in earlier life few had been more active in propagating his opinions, or had incurred more active opposition in doing so. Dr. Bennet received the greater part of his medical education in Paris, where he served as *interne* under Velpeau, Gendrin, Jobert de Lamballe, Lisfranc, and other distinguished men. Returning to London about 1843, he became obstetric physician to the Royal Free Hospital, and took a leading part in introducing the speculum into general use, a task in which he and those who thought with him, encountered, not only professional dissent and opposition but even grave imputations on their personal character. All that has now, after the lapse of nearly half a century, passed away, and

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the use of the speculum is seen to be a necessity for the scientific practice of obstetrics, however grave we may admit the evils to have been which have followed from its abuse by unscrupulous men. Dr. Bennet was also for many years one of the editors of the 'Lancet.' He early became a Fellow of this Society, but did not contribute to our 'Transactions.' His chief work on "Uterine and Ovarian Inflammations," passed through four editions in England and five in America, and was translated into French and German. When compelled, by the cause above mentioned to make his home abroad, he betook himself to Mentone, where he spent the winter, passing the summer at his country house near Weybridge. He was the chief agent in the establishment of Mentone as a health resort, and his work on the 'Treatment of Pulmonary Consumption by Hygiene, Climate, and Medicine' is one of acknowledged value on a subject which can never lose its vital interest for our countrymen, and of great importance for those who study the climate of the Mediterranean.

In 1887 he retired from active life, and thenceforward he resided chiefly in Italy. Here he enjoyed what "he described in his own picturesque style as 'mentally a geological denudation with the reappearance of early sympathies, literary and artistic, obscured by a long and arduous professional career.' To the end of his life his sympathies were warm, his intellect clear, and his affections tenacious. His frequent reappearances at the annual meetings of the British Medical Association were always greeted with pleasure by a host of friends to whom he had endeared himself not less by his intellectual qualities than by his social kindness and amiability. He was hospitable, independent, serviceable to all, and without a tinge of bitterness, although he had encountered severe and even unfair opposition in the earlier part of his career."

So writes a well-informed and friendly biographer, and it is a pleasant description of a life which had been active, energetic, even stormy enough, but which closed in peace and general good-will.

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Dr. Robert Hunter Semple was a well-known and much-respected physician in London. He joined our Society in 1875, and in 1879 served on the Scientific Committee which investigated the relations of croup and diphtheria,[1] a subject on which he had worked much and well, and on which he had published several original works, besides editing some translated 'Memoirs on Diphtheria' for the New Sydenham Society in 1859. Dr. Semple was the author of several other works also, on 'Diseases of the Heart,' on 'Cough, its Varieties, Causes, and Treatment,' and on 'Diseases of the Nervous System,' besides numerous contributions to the journals, of one of which he was for some time editor.

Dr. Semple had for some years been suffering from sclerosis of the spinal cord, before he succumbed to a fit of cerebral apoplexy.

Dr. Hugh J. Sanderson, late of Upper Berkeley Street, died suddenly on August 5th, at Brighton, from heart disease. He was an old member of our Society, his election dating from 1849, and he had served the Society as a Member of the Library Committee in 1862–3, and on the Council in 1872–3; but he did not contribute to our 'Transactions,' nor did he leave any writings. He was born in 1821, became qualified in 1842, at the earliest legal age, having received his education at Liverpool and at St. Bartholomew's Hospital, and took the degree of M.D. at St. Andrew's University in 1853, and that of Member of the Royal College of Physicians in 1859. He soon entered into practice, and afterwards joined the staff of the Blenheim Free Dispensary and the Hospital for Women. Dr. Sanderson was a man of easy fortune and neither gained nor sought a large practice. He was of a singularly retiring disposition, and lived a quiet, uneventful life, though he was most genial and sociable when in company with his intimate friends. He was extremely kind-hearted, and much of his practice was of an unremunerative nature. He was a regular, but silent, attendant at most of the

[1] See their report in vol. lxii of our 'Transactions.'

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medical societies, till failing health prevented him from going out into the night air.

Mr. Samuel Cartwright, who died on August 23rd, at the age of seventy-five, was, like his father, one of the chief men in the dental profession, and inherited also his father's love for art and artists, and for the society of men of letters. He had received his education at Trinity College, Cambridge, and at the London Hospital, before he succeeded to his father's well-known and long-established practice in Old Burlington Street. He was not only well versed in his own and other branches of his profession, but had a great love of art and music, and was well skilled in all matters relating to the drama. In his youth he had been intimate with "Jack Bannister," and had learned the elements of acting from that famous comedian, and he loved to take a part in theatrical entertainments. In the pictorial arts too he was a good critic and no mean amateur. His kindly disposition and his thorough knowledge of the world rendered him a most valuable friend and adviser, and few men were more widely respected and beloved. To his own branch of the profession he rendered the important service of being instrumental in obtaining its recognition at the College of Surgeons by the establishment of the dental examination. He served on the Council of this Society in 1860–1, and on the Scientific Committee on Chloroform in 1863. For some time before his death his health and vigour had been failing, and he "had practically retired from all active work" for some years.

Dr. Barker, formerly of St. Thomas's Hospital, died on October 19th last, at the age of eighty-four. His name had stood as the senior on our *rôle* of Resident Fellows for several years. He had retired from active life and had for many years been enjoying a serene old age; but at one time he was active and zealous in professional research and in the work of his hospital and school. He graduated as M.D. at Edinburgh in 1829, and then after a period of study at Paris and Dublin he resided at Cambridge and took the degree of M.D. there. Dr. Barker joined this

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Society early, his name appearing in the list for 1833. He became Assistant Physician at St. Thomas's Hospital in 1839 and full Physician in 1840. This appointment he held during twenty-eight years, and on his retirement in 1868 the Governors of the Hospital marked their sense of his great services by making him an Honorary Governor. He had given no less efficient services to the school over which he for some time presided as Dean. Dr. Barker was an active Fellow of this Society, having served for two years each in the offices of Councillor and Vice-President, and as Treasurer for three years. His contributions to the 'Transactions' were of considerable clinical interest. The first (in vol. xxviii) is a very good account of two cases in which aneurysms existed, and which were carefully examined with a view to detect that disease, but in which neither pulsation nor bruit could be perceived, a circumstance which does undoubtedly occur in some exceptional cases, and of which I have endeavoured to give an account in a paper published in 1875 in vol. vii of the 'St. George's Hospital Reports.' "Laryngeal diseases and the operation of tracheotomy for their treatment," form the subject of two very thoughtful papers in vols. xxxi and xxxviii—papers which are not, of course, now of the practical value that they were so many years since, but which are interesting as showing the difficulties of the subject before the introduction of laryngoscopy, and, in spite of this, the general correctness of the conclusions to which a man of trained skill in diagnosis, of acute mind and accurate observation, could attain. Dr. Barker's advocacy of the operation of tracheotomy is most judicious, and his opinion to the following effect is I believe still true: "For one case where the operation may be performed prematurely or unnecessarily, I believe it to be neglected in many where it offers a fair prospect of prolonging or saving life" (xxxviii, p. 229). Dr. Barker's other papers are, one on the difficulties which sometimes attend the diagnosis of pleuritic effusions (vol. xxxi), and an interesting description (in vol. xlviii) of that somewhat rare malformation in which

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the aorta is obliterated just below the junction of the ductus arteriosus. In this instance the arch of the aorta (or the artery on the cardiac side of the obstruction) had become aneurysmal and had given way, causing fatal hæmorrhage into the cavity of the pericardium and the substance of the heart.[1]

Such is the nature of a very valuable series of papers, one which well illustrates the practical nature of much of our 'Transactions,' and the influence they have had on the progress of medicine in this country and abroad.

The evening of Dr. Barker's life was a quiet and happy one, cheered beyond that of most men by the affections of this life and the hope of a better.

Mr. John Morgan, who died after a short illness on November 20th, was one of the most eminent and respected practitioners in the west of London, where he enjoyed an extensive practice and numbered many of the foremost men of the time amongst his patients. And so much did his sterling qualities and his genial kindness endear him to those with whom he came into contact that his patients almost universally became his friends. He was successful not only in his medical practice—and there were few better practitioners—but also in all those offices of a friend and a citizen which keep a man's memory fresh long after his life's action is over.

Mr. Morgan was a native of Bath, and was in his seventy-second year at the time of his death. His father was also a medical practitioner, but died in his son's infancy. Mr. Morgan was educated at King's College School, and was then apprenticed to a gentleman who practised near Dorset Square, and entered as a student of St. George's Hospital, where he gained several prizes; amongst others the Brodie Medal for Clinical Surgery, which was obtained thirty years later by his son, who is now an active and distinguished Fellow of the Society.

[1] If the number given in the list of Fellows is correct, there should be a sixth paper by Dr. Barker in the 'Transactions;' but I have been unable to find it.

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Mr. Morgan settled in the neighbourhood of Hyde Park Square, where he soon obtained an extensive practice. His success was due not less to his sagacity and resources in emergency than to the charm of his manner and the kindness of his heart. In middle life he was almost overwhelmed by the demands on his time, which only his strong physique and excellent health could have enabled him to meet. Later on, he obtained some relief by taking a partner, and was thus enabled to enjoy frequent holidays. These he delighted to spend either in foreign travel, or in the country pursuits to which an active boyhood had habituated him. He was a working member of this Society since 1857, having served on the Library Committee in 1862–3 and in the Council in 1880–1; and he contributed a paper to our 'Transactions' (vol. xlviii, p. 39), which was valuable as an additional example of a somewhat rare disease, simple ulceration of the bowel communicating with the bladder, and which had an added interest for myself, as a similar case occurred to me in the following year and was temporarily relieved by colotomy (vols. xlix and l).

Mr. Morgan's vigorous health and active habits seemed to promise a long and healthy old age, but "an attack of influenza last spring left consequences which he had barely thrown off when the acute attack of inflammation in the throat occurred which proved fatal in the course of a week." The numerous assembly which gathered soon after his death, under the presidency of the Chief Justice of England, to consider what form a memorial of him could best take, testified to the esteem and respect in which he was held; and their decision to commemorate him in connection with the Medical Benevolent College, in which he took so warm an interest was universally approved. But no memorial, beyond his own sterling worth, is needed to keep him in the minds of those who have benefited by his kind and sagacious advice, as I myself have often done.

Dr. G. Kenmure Hardie, who died suddenly on Decem-

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ber 26th, was born at Manchester on January 18th, 1823. He graduated at Edinburgh, and entered at an early age into the Army Medical Service, in which he rose to high rank by hard and honourable service. He was appointed Assistant Surgeon to the 53rd foot in February, 1847, and served with that regiment in the Punjab campaign in 1849. Here he was present at the battle of Goojerat, and received the medal and clasp. Dr. Hardie saw much foreign service in the East Indies, Mauritius, China, and Suez, and rose gradually through the various ranks of the Army Medical Service till he retired with the honorary rank of Deputy Inspector-General in 1872. I have the highest authority in his branch of the service for saying that "he was an able, zealous, and efficient medical officer, highly esteemed by his brother officers and by all who knew him, and possessed attainments of a very high order both in professional matters and in general literature. He was an excellent scholar, and was well versed in philosophy and metaphysics; and he was in every sense a thorough gentleman, and a credit to the Army Medical Department."

Mr. John Wood, of King's College Hospital, died on December 29th, at the age of sixty-six. He had long been in failing health, and death did not come till his vigorous intellect had given place almost to unconsciousness, and his restless activity to nearly complete paralysis. It was not without a hard struggle that he recognised the absolute impossibility of continuing the active life that was dear to him, but for the last three years of his life he had been quite laid aside, and his friends watched with helpless pity the gradual increase of spinal paralysis spreading slowly to the brain and gradually obscuring memory and perception. So long, however, as he retained command of his intellect he was cheerful and interested in the affairs of his profession and his friends, and the abundant affection of his home circle did all that could be done to soothe his passage to the grave.

He was a native of Bradford, and retained to the last

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the (to me familiar) accent of Yorkshire. Originally it is said that he was destined for the law, but he was laid up for some weeks by the accident which rendered him lame for life, and during his confinement to the house something induced him to change his profession. That accident caused a dislocation of the hip, which remained unreduced. Having elected to follow the medical profession he became apprentice to a medical man at Bradford, then came to London, and entered the Medical Department of King's College in 1846. His student career gave ample promise of his future eminence, and he obtained the double qualification as early as possible, and then became Sir W. Fergusson's house surgeon in 1850. Afterwards he obtained the appointment of Demonstrator of Anatomy, under Mr. Partridge as professor, an appointment he held for twenty years, and to which he owed his profound and accurate knowledge of the scientific basis of surgery. The preparation of his Jacksonian Prize Essay on Hernia no doubt turned his attention to the subject which he made so specially his own, viz. the radical cure of hernia, and which he further explained to the profession in the elaborate paper contained in the 43rd vol. of our 'Transactions' (1860), read shortly before the appearance of that essay. This operation was, I believe, entirely original, and it was certainly a very great improvement on those then in use for the same purpose (Bonnet's, Gerdy's, Wurzer's, Rothemunde's, &c.), all of which I believe to have been entirely illusory or productive of temporary benefit only. This certainly cannot be said of Wood's operation. In the skilful hands of its inventor it was extensively used, and its permanent success in a large percentage of cases was conclusively proved by Mr. Wood when he lectured on the subject at the Royal College of Surgeons in the year 1885. There can, I think, be no question that the invention of "Wood's operation" for the radical cure of hernia was a great step forward in surgery, equally creditable to its author's anatomical knowledge and surgical ingenuity. That the great pro-

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gress made recently in the method of performance and in the after-treatment of surgical operations has rendered the simpler and more certain "open method" feasible, nay, almost free from danger, is true enough, but in no way detracts from Wood's merit. Besides this admirable paper Mr. Wood contributed two others to our 'Transactions,' one in vol. xlvi, a very interesting account of a complicated operation for the removal of the cicatrix of an extensive burn of the neck and forearm, and its replacement by skin transplanted from remote and adjacent parts of the body; and a third in vol. lii, on the deformity called ectopia vesicæ, and on an operation for its relief much resembling in its main features one which I had previously employed (as stated by Mr. Wood) at the Children's Hospital in the year 1863. Besides these valuable contributions to our 'Transactions' Mr. Wood rendered other services to the Society as Referee of Papers, on the Library Committee, on the Council for 1867–8, and as Vice-President in 1877–8.

Mr. Wood was a man who earned the universal respect and esteem of the profession, and as a friend truly says of him in the public papers: "His memory will be cherished by his numerous band of friends and pupils, to whom he was endeared by his capacity and humour, by his sense of honour and rectitude, and by his friendly disposition. His zeal as a teacher and his original contributions to surgery will form an even more enduring memorial."

Mr. Wood's public position was an eminent one. He was long a Member of the Court of Examiners at the College of Surgeons, and a most painstaking, impartial, and efficient examiner. He also served for many years on the Council and became Vice-President before his resignation of his seat on the Council. He had been Examiner in Anatomy at the University of London and in Surgery at the University of Cambridge and the College of Physicians. His worth was the more conspicuous and the more appreciated as he rose to higher positions—and those who, like myself, had long been his colleagues, and were

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privileged to call themselves his friends, were best qualified to declare it.

Mr. Berkeley Hill died on January 7th last, unexpectedly to his friends, who were used to look upon him as one of the most vigorous and active of men, and to predict for him the same length of days as was attained by his eminent father, the late Recorder of Birmingham, and his still more famous uncle, the introducer of penny postage. The rapid development, however, of internal cancer cut short his career in the middle of its activity.

Mr. Hill was educated at the school of Bruce Castle, Tottenham, so long presided over by members of his family, and then took his M.B. degree at the London University in 1859, obtaining a gold medal in surgery. Soon after this he took the diploma of Fellow at the Royal College of Surgeons, and became House Surgeon at the Children's Hospital in Great Ormond Street, where I first had the opportunity of knowing him and appreciating his worth, as I was then Surgeon to that hospital. He then went abroad for a considerable length of time, and it was from the teaching of Ricord at Paris that he first derived that intimate knowledge of venereal affections which he shortly afterwards turned to such good account. Returning to England in 1862 he soon connected himself as Assistant Surgeon with University College Hospital, and afterwards became Surgeon to the Lock Hospital. In 1864 he began to take part in the movement which led to the passing of the Contagious Diseases Acts, of the policy of which he was an ardent supporter. He was one of the secretaries to the association for extending those Acts; and it is said that at one time he even contemplated giving up practice altogether, in order to devote himself entirely to the task of putting a stop to the ravages which the unchecked spread of syphilis causes amongst the unfortunate population who suffer for the sins of their fathers. However that might be, it is certain that he was one of the weightiest advocates of a system which, though it was ultimately sacrificed to appeals to popular sentimentality,

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certainly produced great benefits whenever it was tried (and not the least to the unfortunate women who were represented as its victims), and which was regretted by the enormous majority of all the medical men who had experience of its working. The work by which he is chiefly known to surgeons is entitled 'Syphilis and Local Contagious Disorders,' and is acknowledged as a standard treatise on the subject. Mr. Hill did much also in other matters connected with the diseases of the genito-urinary organs. He became one of the chief authorities on stricture, and was an ardent, but judicious, patron of internal urethrotomy. His lectures at the College of Surgeons, on "Endoscopy," recalled the attention of the profession to a subject which will, I believe, one day become of prime importance in practice. His only contribution to our 'Transactions' was an interesting and singularly complete paper (vol. lxv) on "The Removal of a Fibrous Tumour from the Female Bladder." He served this Society well and had filled almost all its offices, having been Referee of Papers, Member of Council, Secretary (from 1881–4), and Vice-President. His knowledge of the Society's affairs and interests was great, and his advice in Council always highly appreciated. He gained high office at the College of Surgeons, being at the time of his death Vice-President and Member of the Court of Examiners.

Few men were regarded with more affectionate esteem than Mr. Berkeley Hill. A friendly writer in the 'Lancet' speaks by no means too strongly in the following lines:

"It was the recognition of his unselfish public spiritedness, in addition to his surgical and scientific eminence, which made doubly certain his election to the Council of the College of Surgeons in 1884. As a councillor or committeeman he was invaluable. His attendance could always be relied upon, and his clear-headedness, attention to business, and impartiality in decisions were certain. Another strong characteristic was tenacity of purpose. Having put his hand to the plough, Berkeley Hill was the last man to look back. His noble attitude with regard to the Con-

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tagious Diseases Acts was the best evidence of this, but it was also abundantly shown in many minor actions. His enthusiasm was easily aroused in what he regarded as a righteous cause, and although his decisions were at times impetuously taken, once arrived at he was not easily turned from them. When it has been said that a man was public-spirited and tenacious of his purpose, it is almost unnecessary to allude to his honesty; and yet in this instance it is well to do so, for Berkeley Hill's honesty was absolute.

"In concluding this review of the moral qualities of the deceased, his kindliness and generosity must hold the place of honour. How many students and professional friends have experienced his goodness and generosity when stricken by illness or misfortune it would be hard to say. Though rather impassive on the surface, he had nevertheless a keenly sensitive nature, apt to be stirred by generous emotions, which evoked in him the neighbourly actions of a true Christian and the hospitable instincts of a thorough Englishman. No greater proof of this could be given than in the tokens of personal regard and of genuine grief shown in the wreaths which at the funeral lay on the coffin. Some of them were from men who, young and careless, were in danger of wrecking their life had not the wise counsel and the timely help of the elder man arrested and turned them back again into the path he himself pursued. The pleasure he felt when such a young man gave evidence that the exertion had not been in vain was keen indeed; but the secret had been so strictly kept that letters of gratitude, written by those who received this friendly help, only now reveal (even to his nearest relatives) what he had done. Of his intellectual endowments, his writings and his high professional status are the best evidence. That Mr. Berkeley Hill was in the very front rank of European syphilographers goes without saying, for his works are read and appreciated wherever surgery is studied. Not only is his great work, 'Syphilis and Local Contagious Disorders,' prepared with characteristic thoroughness and great clinical experience, accepted

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as a sound and exhaustive text-book, but it will be acknowledged that Berkeley Hill's enterprise and industry have done much to advance the surgery of the genito-urinary system in this country."

Mr. Hill's premature death was a great loss to the whole profession, and to this Society in particular.

Dr. Thomas B. Christie, late Medical Superintendent of the Royal India Asylum at Earlswood, was a student of the London Hospital, and a warm friend and supporter of his own school, greatly beloved by his colleagues and fellow students there. He had been a non-resident Fellow of this Society for twenty years, but had not contributed to its 'Transactions.'[1] His services were especially given to the institution where he resided, a house historical as the residence of Spencer Percival, and he had held other similar appointments at Clifton, York, and Hackney. He contributed occasional papers to the medical journals and the 'Journal of Mental Science.' He was a genial, sociable man, and had a very large circle of friends by whom he was held in high estimation. For the last two or three years he had been in very precarious health, and had had a previous stroke of apoplexy three years before his death, which took place on January 15th last.

Sir Oscar Clayton died at a good old age on the 26th of January last. He had not done much for this Society (though he served some years since on its Council) nor for medical literature, but he was very successful in practice, had obtained the confidence and friendship of many members of the Royal Family, and received from many quarters, as well as from the Sovereign, marks of distinction and recognition.

My own acquaintance with him does not enable me to speak of him as he no doubt deserved, so I will quote what is said of him by a friend in the 'British Medical Journal.'

"Mr. Clayton had for many years been the personal attendant of the younger members of the Royal Family,

[1] A paper in the 'Transactions' for 1868 is erroneously attributed to him in the 'Medical Directory.'

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and was knighted in recognition of his services. He was very sincerely devoted to his patients, and thought no trouble or exertion too much to fulfil his duties, and to testify his obligation for their trust. He showed great sagacity, and was actively useful on the occasion of the illness of H.R.H. the Prince of Wales from typhoid. He was quick to detect the earliest symptoms, and prompt and energetic in summoning the most highly skilled aid. It will be remembered by those who recall the stirring incidents of those moving times that the first intimation of the probable causation of that attack was made to us by Mr. Clayton. His extensive practice in the fashionable world brought under his care simultaneously not only the Prince of Wales, but also other persons who had belonged to the house party which had assembled to meet him at Scarborough. The relation of these cases he worked out rapidly, energetically, and effectively.

"The lesson thus taught was of great national benefit, and gave a considerable impulse to the life-saving sanitary movement. Throughout this matter Mr. Clayton showed great moral courage, independence of character, and sagacious persistence. His part in these historical events deserves to be long remembered to his credit.

"Otherwise, it may be said of him that while a courtier as well as a surgeon, and much attached to the social conditions of his career as a successful practitioner, he was greatly interested in the Middlesex Hospital, where he was educated, and was always willing and even anxious to give any service to the profession which lay within his power."

Dr. Frederick Leighton, formerly of Bath, the father of the great artist who so worthily fills the chair of President of the Royal Academy, was one of the oldest Fellows of the Society, having joined it in the year 1836. He died a few weeks since in his ninety-third year. His father was a medical man, a Roman Catholic, and resident for a time at St. Petersburg. Dr. Leighton, accordingly, was educated at Stonyhurst, and then went to join his parents at

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St. Petersburg, where his father occupied the position of medical attendant to the Empress. Here he was at first in a merchant's office, but he soon abandoned mercantile pursuits, adopted his father's profession, and obtained distinguished honours in medicine at the University of St. Petersburg. I have been favoured with the perusal of an interesting letter from Sir A. Crighton, then Physician to the Emperor of Russia, testifying to the high distinction which he obtained in this examination. He then returned to this country, and took the degree of M.D. at Edinburgh. He seems to have had a great facility in acquiring languages, for he passed his examination in Russian after only three months' study, and he was equally ready in assimilating technical knowledge, as he used to say that in organic chemistry he had mastered all that was then known before taking his degree. It is true that he used this as an illustration of the little that was then known rather than of his own talent in learning. From Edinburgh he came to London, married in his twenty-seventh year, and then returned to St. Petersburg, where he practised with success for two or three years, when his parents left Russia, and he returned with them to England. He lived for a time at Scarborough, and then set up in practice in London, and joined this Society as a Resident Fellow in 1836. His name, however, disappears from the London list about the year 1841. His practice is said to have been successful, but it was cut short by the invasion and increase of deafness, which compelled him to abandon the profession. He resided abroad from the year 1843, when his father died, to 1852, and from 1852 to 1868 at Bath, when he returned to London. He was, however, little seen in general society, as his deafness had by this time prevented him from joining in mixed conversation. "His one resource," says one who knew him well, "was reading, and he read in many languages and on many subjects. He was for a great part of his life absorbed in the study of metaphysics, but ended in the conviction that it was a fascinating waste of time."

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Dr. Hickson, of Norland Square, had joined the Society only in 1890. He died on January 31st, at the early age of forty-two, from influenza combined with double pneumonia. Dr. Hickson was born and educated in Ireland, and graduated both in Arts and Medicine at Trinity College, Dublin, in 1872. He first practised as a physician, and served the dispensary district of Maryborough, in Queen's County, and subsequently became Assistant Physician to the Birkenhead Infirmary; but afterwards he took up the study of insanity, and resided for fourteen years as assistant medical superintendent in a large county asylum. Here he was "beloved by all for the efficiency, courtesy, and kindness which always distinguished his official and personal relations."

Dr. Hickson only settled in London in the same year (1890) in which he joined the Society. His history and the testimony of his friends leave little doubt that, had his life been spared, he would have been as successful in London as he had been in other fields of practice.

Mr. Sympson, of Lincoln, who died three weeks ago, unexpectedly, from disease of the heart in his sixty-seventh year, was one of the best known provincial surgeons, one of the most eager supporters of every movement that promised benefit either to the medical art or to the medical profession, and one of the most ardent students and practitioners of surgery. His intelligent co-operation was certain to be bestowed on all public assemblages for the promotion of these objects, and no figure was more familiar than his at the meetings of the British Medical Association, or at any of the numerous congresses in the metropolis or elsewhere. He spared no trouble to attend at these gatherings, and though a busy man, and distinguished in his native city, he managed always to find the time for a labour in which he delighted. It was, indeed, to him a labour of love, for he had no desire for professional gain or for personal ostentation; he was animated wholly by a pure love for his profession and for its objects.

Mr. Sympson was a native of Lincoln, where his family

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have been settled for more than a century; and he delighted to dispense a genial and graceful hospitality to his professional brethren who came there, and to show them the wonders of the magnificent cathedral and the many interesting relics to be found in that ancient city. It was in this way that my own acquaintance with him commenced, an acquaintance that soon ripened into friendship.

Mr. Sympson was a distinguished student of St. Bartholomew's Hospital, a dresser for Sir W. Lawrence, and a pupil of Sir James Paget. The latter has said of him,[1] "He was one of my oldest and very best of pupils, admirable alike in study and in conduct, and with this beginning the whole course of his professional life was exactly consistent; it was always and in all things good." With such an introduction to practical life, it is no wonder that Mr. Sympson's bent was mainly to surgery. He served first as House Surgeon and then as Surgeon to the Lincoln County Hospital, an appointment he held till his death; and the hospital owes in no slight degree to his sagacity and support the success of the movement for its rebuilding, which has converted one of the worst into one of the best of country hospitals. So well did Mr. Sympson keep abreast of the surgical progress of his day, that when in 1872 he felt a desire to take the higher diploma of Fellow of the College of Surgeons he was able to pass the examination without sparing a single day from his own busy practice—a striking testimony not only to his own vigour and surgical accomplishments, but also (if an old examiner may be pardoned for saying so) to the practical character of an examination which, though severe enough (and on the day on which Mr. Sympson passed the great majority of the candidates failed), yet need never be dreaded by a good practical surgeon. Mr. Sympson was fond also of the natural sciences, and was a very ardent and intelligent traveller.

"He suffered from influenza in 1890, and was prostrated

[1] 'Brit. Med. Journ.,' Feb. 20, 1892, p. 416.

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by the disease for a time; but a trip across the Atlantic restored him to health, and when he attended the last annual meeting of the British Medical Association he appeared to be as well and as vigorous as ever."[1] Symptoms of angina pectoris came on shortly before his death, and proved rapidly fatal. He leaves a son, also a Fellow of our Society, to continue his father's and grandfather's practice in the old city.

The last on our roll of deaths is *Dr. Edmund Sparshall Willett*, who died at Gunnersbury House, Isleworth, on February 20th, 1892, æt. 64.

Dr. Willett studied at Guy's Hospital and the Bristol Medical School. At the outbreak of the Crimean war he joined the medical staff of the Army, and sailed with his regiment (the 65th Foot) for the Crimea at the end of 1855. He was, however, invalided home, being attacked with fever at Malta, and thus his military career was cut short.

After leaving the army Dr. Willett devoted himself to the subject of lunacy, and in 1856 became the resident proprietor of Wyke House, the well-known private asylum near Brentford, his connection with which was only severed by death.

Dr. Willett was very widely known and greatly respected, the best testimony of which is the fact that he had been a magistrate for his county for many years, and was chosen by the County Council of Middlesex to be one of its Aldermen. Dr. Willett was a gentleman of the best English type, kind, generous, and hospitable; and his asylum was always managed with all the traditional hospitality of a comfortable country house, where the comfort of the guests is the first consideration of the host. He was an ardent sportsman, and, while his health permitted, hunted regularly. As a magistrate he tempered justice with mercy, and his poorer neighbours had in him a rare friend, ever ready to help them with good counsel or pecuniary aid.

[1] 'Brit. Med. Journ.'

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Turning from this record of the friends and fellow-labourers whom we have lost to the present condition of our Society, I am happy to find ground for nothing but congratulation.

The best way to judge of the internal history of the Society, and the actual condition of its finances and of its progress, is to compare the estimate of the income and expenditure for the year 1891, given in the Report of the Building Committee, which was amended and adopted by the Council on February 5th, 1891,[1] with the actual facts shown in the Treasurer's accounts, as audited and now laid before the Society.

The Committee estimated for an income of £4006, composed of subscriptions, fees, &c., £1700, and rents £2306. The sum actually received has been—

Subscriptions, fees, &c. . .	£1733 8 11[2]
Rents	2646 6 3
Interest on Permanent Endowment Fund	12 14 6
Total income[3] .	£4392 9 8

The rents are in excess of the sums given in the final report of the Building Committee, included in the present report of the Council. This shows a total of £2331. But the report of the Building Committee referred only to the permanent tenants. It did not comprise such items as the 100 guineas received from the Congress of Hygiene, nor any other sums received for the temporary use of the meeting-rooms. The meeting-rooms produced in the year

[1] 'Transactions,' vol. lxxiv, p. xciv.

[2] Thus stated in the account:

Subscriptions and Fees . . .	£1686 9 0
Sale of 'Transactions' and 'Proceedings' .	44 2 11
Miscellaneous	2 17 0

[3] Exclusive of a sum received from the Prudential Insurance Company, which is about balanced by a payment on the other side. Neither of these sums will figure in future years (*see* footnote, p. 38).

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1891 £112 3*s.* 6*d.*; the rest of the excess over the sum stated in the estimate, *i. e.* about £200, was made up of arrears or balances due for the year 1890. Such arrears do not, I believe, exist in the account for 1891. On the other hand, the permanent rents have been lately increased; an additional room is in the course of construction by the conversion of the lavatory on the staircase between the ground and first floor, which we could let now at a rent of £40, but which, if combined with use of meeting-rooms, will probably produce more; and I am glad to say that the letting of the meeting-rooms for occasional meetings is producing much more this year than the very small sum which was all that was realised last year beyond that received from the Congress of Hygiene.

So much for the estimated and realised receipts of last year. I believe that those of the present year will be little, if at all, below those of 1891. We shall not, thanks to the care of our Treasurers, have any arrears to recoup, but we shall have an increase on the permanent rents, on the fees and subscriptions, and, I hope, on the occasional use of the meeting-rooms.

On the side of "Expenditure" the Committee estimated "rent, rates and taxes" at £232, and the water rate at £50. The sum actually expended was £229 11*s.* 8*d.*, including the water rate.[1] "Electric lighting, coals and gas" stand in the estimate for £240, and in the account the item "lighting, cleaning, and heating" comes out as £298 14*s.* 9*d.*[2] Furniture and repairs were estimated at £40, and cost £37 2*s.* 10*d.* The meeting expenses were below the estimate, £20 5*s.* 4*d.* instead of £40. The sum spent on the library, £439 18*s.* 7*d.*, considerably exceeded the estimate (£300). This excess was due to large arrears which remained over from the previous year, when the bookseller's account had not been rendered in a satisfactory manner. The sum expended on the year was therefore really below the estimate. Yet, if it had been above it, I

[1] The water rate is now settled at £30.

[2] Extra heating has been found necessary in the library and corridor.

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think no Fellow of the Society will dissent from the opinion that I, for one, entertain that no money could be spent better.

The estimate for "Transactions and Proceedings" was £350, the actual expense £321 8*s*. 7*d*. "Stationery and printing" was considerably above the estimate, viz. £122 2*s*., instead of £80. The excess was occasioned by the necessity of issuing numerous expensive circulars in reference to the debentures, &c., and by the reprinting of the Bye-laws. These are charges which will not recur.

"Salaries and wages" show an excess over the estimate of £67 18*s*. 6*d*., caused partly by the necessity of employing more labour in keeping the premises in order than had been provided for in the estimate, and partly by expenditure on type-writing. A saving in the latter item has been made from the beginning of the current year.

The "miscellaneous incidental expenses," which were estimated at £100, stand in the account as £124 1*s*. 6*d*.[1] To this trifling excess is to be added the expense of the very successful conversazione held last June, which had not been in contemplation when the Committee reported; this amounted to £149 3*s*. 9*d*. The Council also thought fit to reward the extra labours of Mr. Coldrey and A. Tapson with gratuities amounting to £30.

The result of the whole year was that the ordinary expenditure, which was estimated at £2132, amounted to £2510 12*s*. 3*d*.

[1] Thus composed:

Fire buckets	£7 6 0
Clocks	43 19 0
Framing portraits	19 8 6
Spas Committee	16 9 7
Stamps, &c.	29 16 6
Miscellaneous	5 12 8
Cheques	1 9 3
	£124 1 6

Several of these items might be classed under "furniture and repairs," but the distinction is unimportant.

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The above details will, I think, fully account for the excess, if indeed that can be called an excess over the estimate which was caused chiefly by expenditure voluntarily undertaken by the Council, and which was justified by the flourishing condition of the Society's receipts.

There are several items of extraordinary expenditure which will not recur in future years, viz. the payment of rates for the Microscopical Society, £29 6*s*. 3*d*.; the annuity of £34 16*s*.,[1] and the payment to the Building Account £200. On the other hand, future accounts will contain two payments of interest on the debentures instead of one. For the current year, I do not believe that the ordinary expenditure will be so great as for the past; and if this should be the case, it will enable us with ease to pay the £1700 which we estimated as the annual sum to be set aside for interest and redemption of debt.

The balance standing to the Society's credit on December 31st, 1891, amounts to the large sum of £1450 9*s*. 11*d*. But from this should be deducted, in estimating our real financial condition, the sum payable next day for interest on the January coupons, since the present account contains only one half-yearly payment of interest. In future years the two payments on January 1st and July 1st will of course appear, and then it will be perfectly easy to compare one year with another. The payment on January 1st, 1892, would not be quite as large as the subsequent half-yearly payments of £720, but there is also comprised in the balance a small sum (£21 2*s*.) on account of unpresented coupons. On the whole, the effective balance carried over to 1892 may be taken at something over £700. It is to be hoped that the Council now to be elected will see their way to pay out of this balance the sum required to bring the yearly payment on account of the debt to the sum named in the Building Committee's report ('Trans,' vol. lxxiv,

[1] This, however, was nearly balanced by a payment from the insurance office. In future the payments will be made to the annuitant direct from the office, the sum thereby gained to the Society having been paid to the Endowment Fund.

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p. xcv, foot-note) of £1700, or perhaps I should say of £260 beyond the interest paid, for the estimate in the 'Transactions' refers to years subsequent to the completion of the bonded debt, and this has only been completed a few days ago. Still, in strictness, only the composition fees (£77 14*s*.) are necessarily payable to the trustees of the debenture-holders under chap. v of the Bye-laws as revised by the last Annual General Meeting.[1]

One thing remains to be explained on these accounts, viz. the fact that the half-yearly interest on the debentures due July 1st, 1891, was paid out of the Building Account, and not out of the general funds of the Society. This is really a mere matter of accounting, and has no influence on the pecuniary position of the Society. It has, I think, been satisfactorily explained in the statement made by the Treasurers in submitting their accounts.

The experience, however, of the past two years shows me plainly that the Society committed an error, though not a very grave one, in keeping (or trying to keep) the receipts and expenditure of the Building Committee in a separate account from the general funds of the Society. It was an arrangement which appeared obvious at the time; but, as things went on, it became more and more clear that if that separation were to be rigidly insisted on, it would saddle the Society with needless expense, for as one fund was often depleted while the other had plenty, it seemed irrational either to borrow money from the bankers at interest in order to put the General Account in funds, or to take up fresh bonds at the same interest in order to put to the Building Account in funds, when there was plenty of money in hand to meet the liability. And I therefore believe that the course taken by the Treasurers, whether strictly correct or no from an accountant's point of view, was the best for the Society. All that can, as far as I see, fairly be said against it is that the Building Account has not yet been audited. It can, however, now easily be audited, since the final payments on that account are all

[1] 'Transactions,' vol. lxxiv, p. cv.

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made, and I confess that my own opinion would be that this account ought to be put into the auditors' hands. But this is a matter for the Council's decision. The letter of the Bye-law (chap. ix, v) seems only to apply in strictness to the annual accounts; but its spirit should surely govern our action in regard to so much larger an expenditure.

I may here note, for the information of the Fellows in general and the bond-holders in particular, that the whole of the bonds are now issued; and that if any Fellow wishes to get rid of his bonds, or, on the contrary, to take over those held by another Fellow, the Resident Librarian keeps a register to facilitate transfers, and the Treasurers or the bondholders' trustees (whichever may be the proper authority) will very probably be able shortly to pay off a few of the bonds, though no "drawing" can take place, *i. e.* no bonds can be cancelled, except by consent, before the year 1894.

I trust the Fellows agree with me in thinking that this is a fairly satisfactory position. It must be recollected that we are not a commercial company, and we neither need nor ought to make such terms with our tenants as would produce a commercial profit. Our object was to offer the advantages of the Society's house to the various tenant societies on such terms as would secure us against loss, and enable the Society at some future time to take an independent position as a great central medical institution; and this, I believe, will be the result of what has now been commenced.

A reference to the Council's Report will show that an unprecedented number of new Fellows have joined,[1] and that the Society is more numerous than it has ever been. On the other hand, the death-roll has been a long one, and two more Fellows (both non-resident) have died since the

[1] It must be remembered that the twelve months contained in the Report of the Council and that in the annual accounts are not the same, the former running from February 28th, 1891, to March 1st, 1892, while the latter period is the civil year. The number of new Fellows is stated at 53 in the Report of Council, while 55 entrance fees are acknowledged in the annual account.

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report was in print, bringing the losses by death and resignation up to thirty.

I will now make a few observations on the changes in the Bye-laws which the Council submits for the sanction of the Society. They are intended to meet the anomalies which I ventured in my last Address to point out as still uncorrected by the changes in the Bye-laws made last year (see 'Trans.,' vol. lxxiv, pp. 27, 28). The first, and I think the most important matter was the limit of non-residence. "Seven miles from the General Post Office," the present limit, is obviously too near now, when travelling facilities have put a man living at Richmond or Harrow practically much nearer Hanover Square than one at Camberwell or Highgate used to be. The Council has therefore suggested a distance within fifteen miles of this house.

And in connection with the status of the non-resident Fellows, it was proved to the satisfaction of the Council that the Society incurred pecuniary loss by the present terms of non-residence. It has been thought right, therefore, to charge future non-resident Fellows a yearly fee of one guinea, and to diminish the entrance fee to three guineas: and in consideration of the annual payment the privilege of taking out one volume at a time from the library is offered to these non-resident Fellows, the postage or carriage being, of course, paid by the Fellow.

It is hoped that these arrangements will increase the income of the Society without diminishing the number of non-residents.

Of course no change is hereby made in the payments or privileges of any actual Fellow.

Another important matter is the rate of composition fees for resident Fellows. The scale in use, whether based on actual computation or no, must have had some reference to the value of an annuity of three guineas at an age assumed to be the average age at joining the Society and at various periods of membership. Now that the value of money has fallen so greatly this value has been greatly raised. So we asked our accountants to calculate for us what would

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be arithmetically correct composition fees at joining and at various periods. They reported accordingly, and the scale so calculated was considered by the Council. Its adoption, however, would have pressed very hardly on senior Fellows, and would have probably deterred many juniors from compounding; and though that result would perhaps have been to the pecuniary benefit of the Society, it was felt that in some cases the opportunity of compounding is regarded as an inducement to enter the Society. Accordingly a compromise between strict commercial fairness and the too indulgent scale now in use is submitted for your approval.

One other real change is proposed, viz. to abolish the restriction on the number of general practitioners. The experience of the Society seems to show that the restriction is useless, since the proportion of general practitioners has not now, in more than eighty years, risen to the limit, and if it should do so it is difficult to see how it could injure the Society.

The other alterations are matters rather of form than substance, and many of them are only consequential on the adoption of the preceding changes.

Another most interesting document before us is the report of the hon. librarians. For the library is, to my mind, almost as distinctive a feature of this Society, and as great a glory to it, as the meetings for discussion and the grand series of 'Transactions' and 'Proceedings' which have sprung out of those meetings. It is in the enlargement of our library, and the increase of all the facilities which we can offer to our Fellows for its use, that the Society will doubtless find the first object on which to expend any surplus left after providing for the debt. It is, therefore, with genuine pleasure that the Fellows will hear that the Library Committee feels itself released from the necessity of economising, and that nearly 200 volumes have been added to the library, exclusive of the series kept up, while nearly 300 volumes and pamphlets have been received. I earnestly hope that this rate of increase

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will be accelerated, and that the new Council will feel itself empowered to devote still larger funds to this most essential part of the Society.

Another matter connected with the library has long seemed to me to press for examination, but hitherto has been deferred for more urgent business. I mean the large space which is, as I believe, occupied by utterly useless books. The Library Committee's report shows that already the space for shelving is found to be not too great, so that the withdrawal of the Royal Historical Society's books from the North Room is felt as a sensible relief. Ought we not, then, to take some practical steps to carry out a direction which was given some time since by the Council, that as soon as possible the collection should be carefully inspected, and a report made as to the books which are either superfluous (as being duplicates of works rarely used) or are, in the judgment of the Committee, worthless?

I may add that there are some obvious wants in our library in the class of books of general reference, such as the 'Encyclopædia Britannica' and the 'Dictionary of Biography,' which it would be well to supply as soon as possible.

Of the other matters which I brought under your notice last year, the one of chief importance has, I am glad to say, been allowed to rest for a time. I mean the re-discussion of the question of amalgamation. As I said then, it seems to me premature to attempt to settle this question "till a few years' experience has tested the financial security and the scientific progress of this Society in its new situation." But I equally hold to the opinion, which I then also expressed, that it "will inevitably be re-discussed." In fact, those of us who believe that our Society has entered on a new phase of its life, and on an era of greater prosperity and resources, believe also that it will develop into a grand central institute or academy of medicine, which will naturally attract to itself and absorb into its own body many if not all of the minor societies, which have for their object the prosecution of one branch

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only of medicine or of the allied sciences. But I, for my part, am willing to leave the conduct of any scheme of the kind in the hands of our successors, satisfied that it will receive intelligent and liberal treatment from them.

One other subject which I ventured to introduce to you was the advisability of holding occasionally meetings of a character less formal than our regular discussions of papers, such as a house dinner or a conversazione. I am glad to say that the Council accepted this suggestion, and on June 23rd last we held a conversazione here, at which it is calculated that about 3000 guests and Fellows assembled. The gathering was a very pleasant and successful one, notwithstanding that the unfortunate weather prevented our making the use of the Square gardens which we had intended, and which would no doubt have been one of the most agreeable features of the entertainment. The cost charged in the accounts was voluntarily sanctioned by the Council in view of the flourishing condition of the Society's affairs, so that though it appears to have caused an excess of expenditure over estimate,[1] it ought fairly to be excluded from any such comparison, as it was not in contemplation at all when the estimate was made.

On that occasion so many invitations were issued to gentlemen that it was found impossible to invite ladies. On some future opportunity (if future Councils should decide to repeat it) other arrangements might be made. At any rate, our first experiment in this direction was, I think, eminently successful.

The other experiment, that of a house dinner, was carried out yesterday, and, I think, with equal success. One hundred Fellows of the Society assembled in this hall, and passed what seemed to me and to all those with whom I had the pleasure of conversing a thoroughly pleasant evening.

I must allow that I attach a good deal of importance to

[1] The actual cost of the conversazione appears as £149; but it also swelled the expenditure on other items of the account, such as postage, framing of portraits, stationery, &c.

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these social gatherings, even in regard to their bearing on the serious work of the Society. Anything which makes it more kindly and more legitimately popular, anything which tends to bring its Fellows closer together and make them more friendly with each other and with the rest of the profession, appears to me to strengthen it, and to encourage all needful improvements in its working, and so I am the more rejoiced at the conspicuous success of these experiments in that direction.

Looked at, then, in every way—financially, scientifically, and socially—I think we may say that our Society has made great and rapid progress since we left Berners Street, and that its future will, in all probability, justify the best anticipations of its Fellows and well-wishers.

And so, gentlemen, I resign into your hands the great trust with which you have honoured me. I have done my best to carry it out; and although conscious of my own shortcomings, I may say that I have received such efficient assistance from my colleagues and from your excellent Resident Librarian that I believe the Society has not suffered in my hands, and will flourish still more in the far more capable hands of my successor.

I am deeply grateful to you for having bestowed on me the greatest honour with which my professional career could have been graced.

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A CASE

OF

LIMITED LESION OF CERTAIN SPINAL NERVES

BY SECONDARY CARCINOMA OF THE VERTEBRÆ AND DURA MATER.

BY

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AND

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Received March 31st—Read October 27th, 1891.

M. D—, a married woman, æt. 44, was admitted into University College Hospital on March 4th, 1889, under the care of Dr. Ringer, to whom we are indebted for his kindness in allowing us to bring the case before the notice of this Society. The patient's illness dated from September, 1888, when she first experienced dull aching pains in the region of both scapulæ. These pains persisted, and about six weeks later the patient discovered that she had lost power in the second finger of the right hand. This was followed by gradual loss of power in the other

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fingers and in the hand, accompanied by progressive muscular wasting. The same changes soon followed in the left hand. At this time the patient also experienced constant pain in both arms, with occasional shooting pain in the same position. She became gradually worse, so that in December, 1888, she took to her bed, and remained there until her admission to the hospital. During January and February, 1889, no improvement occurred in the severity of the pain in both shoulders and arms, whilst the muscular wasting and loss of power steadily progressed to the forearms and arms. No further symptoms developed. It may be stated that the patient's previous health had been good, and there was no evidence of syphilitic taint.

The condition of the patient when admitted to the hospital in March, 1889, was as follows :—She constantly assumed the dorsal decubitus, and her face wore an anxious expression indicative of long-continued pain. Her mental condition was clear, and there were no symptoms of cerebral disease. She complained of continuous aching pain in both infra-spinous fossæ, and in the interscapular region at the same level, the pain extending down the arms ; also of shooting pains in the upper limbs, more marked below than above the elbows. The distribution of the pain in the upper limbs was diffused, and bore no apparent relation to the course of the nerves. There was no tenderness over the infra-spinous fossæ, but slight tenderness over the spines of the vertebræ at this level. Pressure was painful along the inner side of each upper arm, and there was some general tenderness over both forearms, most marked, however, along the line of the median nerves. There was no anæsthesia, and no loss of muscular sense. The patient could readily appreciate painful and simple tactile impressions, and the painful parts were especially sensitive to cold.

Condition of the muscles.—There was considerable wasting of the *supra-spinatus* and *infra-spinatus* muscles on each side. When the arms were raised the upper fibres

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of the *trapezius* contracted, and they were not atrophied; the lowest fibres of the muscle for the width of one inch stood out when the arms were passively raised; the intermediate part of the muscle on each side was paralysed and atrophied. The *levator anguli scapulæ* and *rhomboids* were normal on each side. The *serratus magnus* contracted with fair strength, but was apparently somewhat wasted. The *latissimus dorsi* was much atrophied. The *deltoid* possessed considerable power, and was not wasted. The sternal and costal fibres of the *pectoralis major* on both sides were much wasted and did not contract, but the clavicular fibres were not wasted and contracted well. The *triceps* was much atrophied, especially on the right side, where the power of extension of the elbow was nearly lost. Each *biceps* muscle acted fairly strongly. There was considerable wasting of the *extensors of the wrist and fingers*, and there remained but slight power of extension of the wrist and none of the fingers. Pronation and supination could be performed feebly. The *supinator longus* was not atrophied, and contracted readily. The *flexors of the wrists and fingers* were atrophied, flexion of the wrist being especially feeble on the right side. All the intrinsic muscles of the hand and thumb were greatly atrophied, and no movement whatever of the thumb or fingers could be executed. All the joints of the fingers were in a position of partial flexion. There was wasting of the *intercostal muscles* in the first three spaces of each side.

It is thus seen that the loss of power and the muscular wasting were nearly symmetrical on the two sides, being in the case of certain muscles only slightly more marked on the right than the left.

The muscular power and sensation in the lower limbs were good; the patient could walk easily. The knee-jerks were normal, and there was no ankle-clonus.

There was no weakness of the muscles of the face or orbits. The pupils were equal, and reacted readily to light and accommodation.

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In the upper and outer quadrant of the right breast was a moderately hard, rounded tumour, as large as a walnut. Its outline was sharply defined, but the growth appeared to be intimately connected with the surrounding breast-tissue. The nipple was not retracted, but was loosely held to the tumour. The latter was unconnected with the skin, and moved freely on the surface of the pectoral muscle. No enlarged glands could be felt in the axilla.

During the first ten days that the patient was under observation no noticeable change occurred; the pains in the upper limbs were severe, but were relieved by morphine and antipyrin.

On March 14th the patient began to suffer from shortness of breath, and had a weak, ineffectual cough.

On the 27th the note states that the pains, especially in the forearms, were very severe. There was distinct fulness in the neck above the right clavicle, and in the region of the thyroid gland. There was slight dulness below the right clavicle. The tumour in the breast was steadily increasing in size.

On the 29th the respiratory power was much weaker, and mucus was evidently accumulating in the lungs. With inspiration the lower chest expanded, but the abdominal wall receded; there was scarcely any movement of the upper part of the chest. With expiration the recti abdominis muscles contracted strongly. The knee-jerks were slightly increased, and slight ankle-clonus could be obtained.

On the 30th the patient was sinking rapidly. The power of flexion of the right wrist-joint had entirely disappeared; both wrists could still be extended. The elbows could be flexed. Enlarged glands were detected in the right axilla.

Death occurred on April 4th, 1889, seventeen days after the patient's admission into the hospital.

Autopsy.—The *spinal cord* was removed in the usual manner. The dura mater was considerably thickened in

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the region of the cervical enlargement, where its anterior surface was strongly adherent to the bodies of the vertebræ. On slitting up the dura mater it was found to be somewhat adherent to the pia mater in the region of the thickening, especially behind. The thickening of the dura mater was almost confined to its anterior half, the posterior half being only slightly thicker than natural. The thickening was most marked opposite the origin of the anterior roots of the first dorsal nerve, where it measured nearly a quarter of an inch. Above and below this point the thickening gradually became less marked, ceasing above opposite the upper limit of the origin of the anterior roots of the sixth cervical nerve. Below, the greater part of the thickening ended opposite the roots of the fourth dorsal nerve, but on the right side a narrow strip extended downwards as far as the origin of the roots of the sixth dorsal nerve. Thus the fibres of the sixth, seventh, and eighth cervical, and the first, second, and third dorsal nerves on both sides were all involved in what on subsequent microscopical examination proved to be a carcinomatous infiltration of the dura mater. On the right side the fourth and fifth dorsal nerves were also partially involved. The outer surface of the infiltrated dura mater was rough and adherent to the posterior surface of the bodies of the vertebræ, which were also infiltrated with new growth; the inner surface of the dura was, however, smooth.

Muscles.—The *supra-spinatus* and *infra-spinatus* were pale in colour and atrophied. The *latissimus dorsi* was very pale and much atrophied. The clavicular fibres of the *pectoralis major* were dark red in colour and normal in bulk, whilst the sternal and costal fibres were pale in colour and much atrophied, presenting a striking contrast with the normal upper part of the muscle. The *pectoralis minor* was pale and wasted. The *biceps* was normal in appearance, but the *triceps* was much atrophied. The *intercostal muscles* of the three upper spaces were pale and wasted; the fourth and fifth appeared to be slightly

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affected, whilst the sixth and lower ones presented a natural appearance. The *trapezius* was normal in appearance, and the paralysed middle part of the muscle was not obviously atrophied.

Breast.—In the upper and outer quadrant of the right breast was situated a rounded tumour as large as a Tangerine orange. In the appearance of its cut surface and in its relations to the surrounding tissues it presented the characters of a carcinoma. The remainder of the breast tissue had a normal appearance. Several axillary lymphatic glands were infiltrated with soft white growth. The *superior mediastinum* was filled by a mass of growth which extended back to the spine and infiltrated the anterior common ligament. The body of the sixth cervical vertebra and the upper part of that of the seventh were very dense and of a yellowish white colour, contrasting markedly with the pink appearance of the others. The intervertebral discs appeared normal.

The left internal jugular and innominate veins were dilated, whilst the right were contracted. At the junction of the two innominate veins a mass of growth projected into their lumen, but the intima remained intact. There was firm adherent clot in the right subclavian and axillary veins as low as the lower border of the subscapularis.

The *left lung* was healthy, the *right* was universally adherent.

The *thyroid gland* was enlarged and infiltrated with nodules of soft white growth, the largest being half an inch in diameter.

Abdomen.—The *intestines* were natural. In the end of the *vermiform appendix* was an oval mass of growth measuring seven eighths of an inch by half an inch. It appeared to have arisen in the subserous tissue, and did not involve the mucous membrane. In the mesentery of the lower ileum were several infiltrated glands, and others lay on the front of the aorta.

The *liver* and *spleen* were normal.

Each *kidney* contained two nodules of soft white growth,

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those in the left being in the substance of the organ, whilst those in the right lay beneath the capsule.

Both *supra-renal capsules* were enlarged and extensively infiltrated with degenerating new growth.

The *right ovary* measured 3¾ by 2¼ inches, and was divided into two unequal parts. The larger consisted of a mass of soft pinkish-white growth, whilst the smaller presented a fine spongy mesh-work, the spaces containing a thin mucous fluid. The Fallopian tube was normal.

The *left ovary* measured 3½ by 2¾ inches; it was infiltrated throughout with new growth. The Fallopian tube was normal.

Brain.—The membranes were natural. At the posterior end of the right gyrus fornicatus was a degenerated mass of growth as large as a small marble, dark in colour, and almost diffluent. It readily separated from the surrounding brain-substance, leaving a smooth-walled cavity. There was very distinct bulging of the cortex of the left hemisphere over the posterior end of the frontal and lower end of the ascending frontal convolutions. On section this was found to be due to a cavity extending to within about half an inch of the surface. It contained a grumous fluid, and was about the size of a small horse-chestnut. It was lined by soft broken-down growth. The growth extended down to the roof of the Sylvian fissure. Another small mass of growth lay three quarters of an inch behind this, and rather deeper in the white matter.

Almost exactly symmetrical growths were found on both sides of the brain in the middle of the internal orbital convolution, and also on the inner parts of the temporo-sphenoidal lobes. A small nodule lay in the white substance beneath the floor of the descending cornu of the right lateral ventricle, and another in the anterior part of the left half of the pons. The left hemisphere of the cerebellum was the seat of five separate nodules of growth, and two nodules lay in the right side.

Microscopic examination.—Sections of the *spinal cord*

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were made at different levels between the origin of the third cervical and third dorsal nerves, and stained with aniline blue-black or carmine. In all the sections examined the grey matter was healthy. There was a tract of slight ascending degeneration in the postero-external and postero-internal columns on each side. On examining the sections from below upwards this tract was first distinctly seen at the level of the roots of the eighth cervical nerve. Here the most distinctly marked part of it lay at the inner side of the posterior cornu for about the middle two fourths of the distance between the posterior commissure and the posterior surface of the cord. The anterior part of the tract extended right across the postero-external column; the posterior part curved outwards with the posterior cornu, and was continued as a narrow strip backwards to the point of entrance of the posterior roots. At the level of the sixth cervical root it was more distinct than in any other section, and reached from the posterior commissure to the posterior surface of the cord. It occupied the inner third of the postero-external column, and was expanded laterally at its anterior and posterior extremities. Just behind the commissure it extended into the postero-median column, of which it involved the anterior third. In the upper cervical region the tract occupied only the anterior fourth of the postero-internal column and the inner half of the anterior third of the postero-external column.

All the organs which were the seat of secondary growths were also examined microscopically, and these growths were found to agree in all essential points with the structure of the primary one in the breast. They were, however, all softer than it, and showed a correspondingly larger proportion of cellular elements, and a greater tendency to degeneration.

Remarks.—Our knowledge of the functions of the roots of the spinal nerves is derived from three sources: experiments on animals, dissections of the human subject, and clinical observation confirmed by post-mortem examina-

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tion. We bring forward this case in the hope that some of the facts observed may help to increase the data of our knowledge derived from this last source of information.

The chief interest of the case arises from the fact that the upper limit of the growth involving the cervical nerve-roots was sharply defined. Thus, whilst the sixth nerve-root was involved, the fifth remained unaffected. We have thus a lesion which closely resembles an experimental one, and such as we do not often have an opportunity of observing in man. This distinct limitation of the lesion is also borne out by the condition of the muscles, which were examined at the post-mortem. The difference between the atrophied and the healthy muscles was very marked, being especially striking in the pectoralis major, where the lower part was pale and atrophied, whilst the clavicular fibres retained their natural colour and appearance. From this we may conclude that the muscles supplied from this part of the spinal cord which remained healthy received at least the greater part of their nervous supply, if not all, from the fifth cervical or a higher root; whilst, on the other hand, the affected muscles were supplied by the sixth cervical and the lower roots which were involved in the lesion. Thus the levator anguli scapulæ, the upper part of the trapezius, rhomboids, deltoid, the clavicular fibres of the pectoralis major, biceps, supinator longus, and supinator brevis were supplied by fibres coming from the spinal cord in the fifth, or possibly higher nerve-roots. On the other hand, the supra-spinatus, infra-spinatus, middle part of trapezius, serratus magnus, sternal and costal fibres of the pectoralis major, pectoralis minor, triceps, extensors of wrist and fingers, flexors of wrist and fingers, latissimus dorsi, the intrinsic muscles of the hand, and the upper three intercostal muscles, received their supply from the sixth, seventh, and eighth cervical, and the first, second, and third dorsal nerves.

We will now compare these facts with the results obtained by Ferrier and Yeo in their experiments on monkeys. In doing this we take the arrangement given in

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their original communication,[1] for in the amended list[2] the small muscles of the hand are given as deriving their nervous supply through the second dorsal nerve, which is not the case in man. Thus the first list, in which the nerve-supply of each group of muscles was subsequently found to be one root too high, is the more useful for comparison. The relation between the two will be most clearly seen in the annexed table. On the left side are the muscles supplied by the various nerve-roots in the monkey, from the fourth cervical to the first dorsal. On the right a cross (+) is placed opposite each of the muscles which was affected in our case. Our notes of the condition of the muscles being not quite complete, the unaffected ones have been marked (−), whilst the condition of those muscles not marked was not ascertained.

Fourth cervical:	
Deltoid	−
Rhomboids	−
Supra-spinatus	+
Infra-spinatus	+
Biceps	−
Brachialis anticus	
Supinator longus	−
Extensors of wrist and fingers	+
Diaphragm	
Fifth cervical:	
Deltoid (clavicular part)	−
Biceps	−
Brachialis anticus	
Serratus magnus	+
Supinator longus	−
Extensors of wrist and fingers	+
Sixth cervical:	
Latissimus dorsi	+
Pectoralis major, sternal and costal	+
„ „ clavicular	−
Serratus magnus	+
Pronators (flexors of wrist)	
Triceps	+

[1] 'Proc. Roy. Soc.,' 1881, vol. xxxii, p. 12.

[2] Ibid., 1883, vol. xxxv, p. 229.

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Seventh cervical:	
Teres major	
Latissimus dorsi	+
Subscapularis	
Pectoralis major, sternal and costal	+
„ „ clavicular	−
Flexors of wrist and fingers	+
Triceps	+
Eighth cervical:	
Long flexors	+
Ulnar flexors of wrist	+
Small muscles of hand	+
Extensors of wrist and fingers	+
Triceps (long head)	+
First dorsal:	
Small muscles of hand	+
Intercostals	+

From this table we see that, as far as the nerve-roots involved in the lesion—the sixth, seventh, and eight cervical and the first dorsal—are concerned, the muscles affected in our case are those which Ferrier and Yeo found to be innervated by these roots. The clavicular fibres of the pectoralis major form an exception which will be considered later. When, however, we consider the fourth and fifth cervical roots, neither of which was involved in the lesion, we find that several muscles were affected which would have escaped had the lesion occurred in the monkey. These muscles are the serratus magnus, the extensors of the wrist and fingers, and the supra- and infra-spinatus. The *serratus magnus* in the monkey receives its nerve-supply from the fifth and sixth roots. In our case the marked atrophy of this muscle indicates that the fifth root took at the most but an unimportant share in its innervation. The *extensors of the wrist and fingers* were affected, and thus were supplied by a lower root than in the monkey. This agrees with Dr. Gowers's opinion[1] that in man these muscles are supplied by the sixth and seventh cervical roots.

The *supra-spinatus* and *infra-spinatus* appear to be

[1] 'Diseases of the Nervous System,' vol. i, p. 129.

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usually supplied by the same root as the other muscles of the group which is generally affected in Erb's "upper arm type" of paralysis, viz. deltoid, biceps, brachialis anticus, and supinators.

Erb considered that paralysis of this group of muscles indicated affection of the sixth cervical nerve-root. Here, however, the supra-spinatus and infra-spinatus were the only muscles of the group supplied by this root, the others being supplied by the fifth or the fourth and fifth together.

Dr. Herringham[1] found from his dissections that the sixth "exercises sometimes an extremely small influence and sometimes none at all over the supra-spinatus and infra-spinatus." According to these observations the nervous supply of these two muscles was exceptional in this case.

The condition of the *pectoralis major* is one of the most interesting points to be considered. In Ferrier and Yeo's list the pectoralis major is given as supplied by the sixth and seventh roots. In our case the atrophied condition of the sternal and costal fibres of the muscle would agree with their innervation from these roots. The clavicular fibres were, however, evidently supplied by a higher root than the sixth, as they were unaffected. We have thus a clear illustration of the double nerve-supply of the pectoralis major. Attention was first drawn to this separate origin of the nerve-supply of the clavicular portion of the pectoralis major by Dr. Beevor,[2] and was illustrated by some cases shown at this Society. Dr. Beevor said, "I certainly think that the pectoralis major may have its two halves assigned to different parts of the cord, the clavicular half going along with the anterior fibres of the deltoid, of which it is a continuation, whilst the sternal half is more associated with the triceps group. This grouping is also illustrated by physiological action, for in pressing the two hands together the clavicular part

[1] 'Proc. Roy. Soc.,' 1886, vol. xli, p. 425.
[2] 'Med.-Chir. Trans.,' 1885.

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of the pectoralis major, the anterior fibres of the deltoid, and the biceps are associated; whilst in pressing the hands downwards against a table the sternal part of the pectoralis major and the triceps act together, the biceps not being used in the movement, which may be illustrated by pressing on the table to assist in rising from a chair." These conclusions receive strong confirmation from the facts observed in our case.

The presence of the tracts of ascending degeneration in the posterior columns of the cord shows that the posterior roots of some of the nerves in the lower cervical and upper dorsal regions must have been compressed by the growth, although at the autopsy it was thought that they were not affected by it. Thus we are unfortunately unable to state from which posterior roots the degenerated fibres came. The posterior roots must have been affected to a much less degree than the anterior, for there was no anæsthesia, and the degeneration is not well marked.

The course taken by the degenerated tract is the same as that which is usually taken by the fibres which enter the cord in the "median bundle" of the posterior roots in the upper dorsal and lower cervical regions.[1]

Similar ascending degeneration was observed by Dr. Howard Tooth[2] in his monkey No. 3, in which Mr. Horsley had divided the posterior roots of the sixth, seventh, and eight cervical and first dorsal nerves, and also in monkey No. 11, in which the left posterior roots of these same nerves and the second dorsal had been divided. In Dr. Tooth's monkey, however, there was also degeneration of the direct cerebellar tract, which was not affected in our case.

Apart from the above considerations the case is of interest as an example of a very wide dissemination of secondary growths in a case of carcinoma.

There can be little room for doubt that the growth in

[1] Foster, 'Text-book of Physiology,' pp. 891—893.

[2] Gulstonian Lectures, 'Brit. Med. Journ.,' April 20th, 1889.

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the breast must be looked upon as the primary one. The growth here was solitary, and the minute structure was that of a glandular carcinoma of the type most commonly arising in the mammary gland tissue; whereas in any of the other situations in which, according to our present knowledge, such a growth could have arisen, the growths were multiple. In a table of 128 cases of general dissemination, compiled by Gross[1] from various sources, in which post-mortem examinations were made, the organs affected in our case were the seat of secondary growths in the following percentage :—axillary glands 89·94, other glands 23·43, bones 7·02, brain 2·34, kidney 3·9, adrenals 1·56, ovary 5·46, and intestine 0·78. The thyroid gland is not mentioned in his list. It is noteworthy that the liver, which is so frequently the seat of secondary deposits in cases of cancer of the breast, had in our case entirely escaped. In cases of carcinoma of the breast the ovaries are perhaps especially liable to be the seat of secondary deposits. Thus Stephen Paget,[2] in an analysis of 735 cases, found one or both ovaries affected in 37, "twice as often as the spleen, and as often as the spleen and kidneys put together."

The remarkable uniformity of structure presented by the growths in such a case as this is one of the most powerful arguments in favour of the commonly accepted view that we have to deal with a single primary growth sowing itself by the lymph- and blood-streams in various parts of the body, and as strongly opposed to any theory which would endeavour to explain such a case by a widespread tendency of the tissues to the development of carcinoma. It is often interesting to note that the exact relations of such secondary nodules serve to distinguish them from a primary growth in the same position. Thus, in the case of the nodule in the end of the vermiform appendix, the mucous membrane was not invaded by the growth, which appeared to have commenced as a subserous

[1] 'Tumours of the Mammary Gland,' 1880, p. 161.

[2] 'Lancet,' 1889, vol. i, p. 571.

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deposit. From it as a fresh centre the lymphatic glands in the lower end of the mesentery had become infected.

We have very scanty knowledge concerning the conditions which influence the position of the secondary deposits of carcinoma. In our case there was a remarkable tendency to bilateral symmetry in their arrangement; thus both kidneys, both adrenals, and both ovaries were affected, whilst in the brain this same tendency was shown to a still greater degree.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 3.)

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A CASE

OF

ACTINOMYCOSIS OF THE VERMIFORM APPENDIX

CAUSING PERITYPHLITIS.

BY

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Received April 8th—Read November 10th, 1891.

J. B—, æt. 50. General health good. Habits active and temperate. No previous illness of note, except, about the middle of 1886, he experienced a slight uneasiness in the right inguinal region, and, fearing hernia, consulted Mr. Bryant. Nothing was found, and he soon got well. The present illness began about the 12th of October, 1888, with some severe, but indefinite and occasional abdominal pains, felt at uncertain intervals, and increased by certain movements of the right leg, such as getting into a dog-cart. He had to take opium twice or thrice, but continued to walk and drive about until October 21st. On that day he was suddenly seized, while sitting at table, with intense pain in the right iliac fossa, so that it was with great difficulty that he could move to the sofa. I saw and

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examined him about an hour after, and, on palpation, felt a distinct, firm, and rather uneven solid mass, very tender to pressure, and in the position of the cæcum. He was believed to have a perityphlitis, and treated by morphia, laxatives only when needed, rest in bed, and bland diet. He improved while resting; the bowels acted painlessly; all the other functions were normal. There was no rise of temperature, and the lump felt in the right iliac fossa diminished in size and tenderness. However, on November 17th and 18th, having walked a little in his bedroom without uneasiness, and even stood upon his right leg without pain, he had an evening temperature of 99·6° F.

November 20th and 21st.—He went downstairs and rested on a sofa. This was followed by an evening rise of temperature to 100·6° F., with an increase of pain, of swelling, and of tenderness in the iliac region. From this date he remained in bed until the end. The swelling soon increased, so that in a few days it involved the whole iliac fossa, and extended above the crest of the ilium, as well as outwards beyond the axillary line. The tenderness was extreme, so that deep palpation was very difficult. The temperature varied much; at times rose to 102·8° F. The pulse 84—90. November 25th.—Was seen in consultation by Dr. P. H. Pye-Smith, whose diagnosis was "typhlitis, with suppuration behind the cæcum." Strict rest and fluid diet were enjoined, and the morphia was continued, but no laxatives were given.

December 5th.—Was suddenly taken with very severe pain in the right iliac region and all down that leg, with a general sense of distress, followed rapidly by venous turgescence, so that in four hours the whole limb was swollen, tender, and livid, with strongly marked superficial veins. Soon this condition was extended to the whole iliac fossa, and all the parts involved were œdematous. Thrombosis of the right external iliac vein was inferred. It is somewhat singular that the temperature, which had been about 100·5° F. for some days, was normal on the 5th and 6th, although it rose again directly after.

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9th.—After a consultation with Dr. Pye-Smith and Mr. C. J. Symonds, an aspirating needle was introduced, with antiseptic precautions and under an anæsthetic, from a point internal to the anterior superior spine of the ilium, in a direction to reach the parts behind the cæcum; but no pus was obtained. There was no subsequent general or local reaction. The treatment was continued as before.

January 4th, 1889.—By this date the œdematous swelling and venous turgescence had almost disappeared from the whole of the right lower extremity and parts above Poupart's ligament, as if the circulation had in some way been restored. It was now again possible to palpate the iliac region, and at this date deep-seated induration was felt from a point near to the anterior end of the eleventh rib down as far as an inch and a half below Poupart's ligament near the great vessels. This area was visibly swollen, and while it was lost without definite boundary outwards towards the flank, it was fairly well defined inwards by a gently curved line convex inwards and forwards. This line appeared to be rather undulating, and also variable according to the states of the gut which bounded it towards the median line. The resistance of the swelling was that of a solid, and so hard, especially near to the crest of the ilium, as to feel like a continuation of the bone. There was but moderate tenderness, and nowhere could fluctuation be detected.

10th.—The swelling and œdema had returned in the right iliac region; the right loin became involved, as well as the buttock and the upper part of the thigh. After a consultation with Dr. Pye-Smith he was treated with Liq. Hydrarg. Perchlor., ʒss ter die. So far, although the skin had been involved in the œdema, and the turgid veins gave a livid colour at times to the parts affected, it had not itself apparently been involved in any truly inflammatory process. About January 24th the skin over the swollen regions was inflamed, a sort of patchy erythema, with redness, induration, and but little tenderness.

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He had more abdominal pain, and more morphia became necessary.

February 14th.—Pointing appeared rather suddenly close to the spot where the aspirating needle had been introduced.

16th.—The swelling burst spontaneously, and yielded a little brownish, offensive, thick pus.

On the 17th Mr. Wright enlarged the opening and let out about 1 oz. of dirty, thick, offensive pus with a fæcal odour. The cavity was explored with a probe and with the finger to the depth of two inches, but no bare bone was felt, nor was any deeper channel found. A drainage-tube of two inches in length was inserted. No anæsthetic was used.

20th.—Was seen by Dr. Pye-Smith, and it was agreed to leave off the mercuric chloride, which had not produced any noticeable effects. After the operation on the 17th the temperature fell to about 100° F.

28th.—After a consultation with Mr. Symonds an anæsthetic was given, a long probe was passed by that gentleman from the existing aperture to the loin, just above the level of the iliac crest, cut down upon there, and with the finger in this last aperture a sinus and cavity were explored in the region behind the cæcum. Although the limits of the cavity could not be reached, Mr. Symonds was able to speak of it as having soft anfractuous walls, yielding in front towards the gut, firm behind towards the muscles, and nowhere feeling hard or nodular or like a growth. A rubber drainage-tube of 5½ inches in length and ½ inch in diameter was introduced from the lumbar opening in the direction of the cæcum, and another rather longer one was passed from the anterior to the posterior aperture. During the operation there escaped some gas, and a moderate amount of fœtid and fæcal-smelling pus, with blood; also some flakes of lymph and of soft granulation tissue were squeezed out of the apertures during the manipulation, and these were preserved in spirit for future examination. The sinuses were syringed out with

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weak carbolic acid solution, and the whole was dressed with mercuric wool; afterwards weak iodine solution was used to syringe the sinuses. The discharges continued free for about a week, and were chiefly from the lumbar aperture. The temperature, which had fallen as above said to 100° F. after the operation of the 17th, fell to normal after that of the 20th, and remained so with but slight fluctuations for nearly a month.

It is noteworthy that up to this date the bowels acted well and easily with enemata, the stools were normal in character and free from pus or blood; the appetite, digestion, respiration, and circulation also normal, and the urine copious and normal in aspect and composition.

The fragments squeezed out during the operation on the 28th, when examined, were found to consist of shreds of coagulated fibrin, of dead connective and fatty tissues infiltrated with leucocytes, and some very vascular granulation tissue. No evidence could be found of any neoplasm. The fœtid and fæcal-smelling discharges contained numerous small, deep brown or blackish specks, which, on examination, were proved to be fæcal masses, mostly of deeply tinged vegetal tissue, and one or two were found having an obscurely crystalline fracture suggestive of a broken concretion. After some days these dark specks ceased to appear, as if a communication with the gut had closed.

March 20th.—The skin having again become inflamed and swollen about midway between the two openings, as if pus were there confined, Mr. Wright made an incision at this point under an anæsthetic, and let out a small quantity of dirty fœtid pus without fæcal odour. No sinus could be traced leading towards the cæcum, but a tube was inserted leading to the lumbar opening, and another to the anterior one.

29th.—The last operation was not followed by any increase of discharge or relief to the local redness and swelling, while the temperature rose to 100° F., and a

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colourless œdema extended to the right foot, the upper half of thigh and hip, as well as to the scrotum.

31st.—Consultation with Dr. Pye-Smith and Mr. Symonds, who withdrew the drainage-tubes last introduced, as they yielded nothing and appeared to irritate. He attempted in vain to reach the deeper cavity from this last made aperture.

April 2nd.—Consultation with Mr. Anderson, who passed a small tube some three inches in the direction of the cæcum from the central aperture, and a little old pus escaped; but the next day this tube was forced out, and could not be introduced.

4th.—As the drainage-tube in the lumbar aperture had been gradually shortened until it was only two inches in length, Mr. Anderson passed a smaller tube from the same aperture more than five inches in the direction of the cavity behind the cæcum, and this tube was gradually replaced by a larger one until a full-sized tube was introduced on April 9th. A tube was also placed between the anterior and the central apertures, but in spite of these apparently sufficient provisions for efficient drainage very little pus escaped, probably not more than was furnished by the walls of the long sinuses. The skin remained red, tender, œdematous, and the temperature was over 100° F. Appetite and sleep were fair, and the patient was hopeful, but he had to take rather freely of morphia and of alcohol.

13th.—The local conditions remaining without improvement, a consultation was again held, and Mr. Symonds under an anæsthetic made a free incision through the thick integuments from the anterior aperture outwards and upwards to the middle one, placed just above and about the middle of the iliac crest. He explored deeply with a probe and with the finger in the direction of the deep focus behind the cæcum to a distance of 5½ inches, and breaking down the soft tissues carefully with the finger he established a free communication with the deep focus from the lumbar and from the anterior openings.

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Into this channel he passed a large fenestrated rubber tube, so as to provide for satisfactory drainage from the deepest cavity and from intermediate sinuses. During the operation there escaped little or no pus, and only a very moderate amount of dark venous blood. Some soft masses of granulation tissue were squeezed out and preserved for examination. After the operation great pain and distress followed, and for some time the temperature rose to 103·4°. For two days there was no other discharge than bloody serum, followed by only a moderate amount of pus tinged with blood, having a rather fœtid but not a fæcal odour.

The discharge contained shreds of dead tissue, flakes of lymph, and some dark brown specks of fæcal matters. It was also now noticed to contain other yellowish, or deep reddish or brownish spherules, having an appearance which reminded one of certain minute ova; these, after careful examination, proved to be the spherules or colonies of actinomyces. I think they could be readily recognised, by any one who has noticed them, by their naked eye characters. If the pus be mixed with water, or the dressings be washed and the fluid be given a movement of rotation, the spherules sink to the bottom, especially the larger and more deeply coloured ones, as quite peculiar bodies. To the naked eye they seem, although variable in size, mostly about the size of a very small pin's head, and appear to have an adhesive slimy or gelatinous envelope with a coloured centre. Under a good pocket lens, and seen as a transparent object with oblique light, the coloured centre appears rather like an irregularly cleaving yelk (see Plate I, Fig. 1). The diagnosis can be readily placed beyond doubt by the employment of a microscope with a moderate magnifying power. The process of preparation need not be complex, and can be carried out at once in the ward. I found it sufficient to place two or three spherules fresh from the washings of the dressings on a slide with a drop of water, covering lightly without greater pressure than the weight of the

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cover-glass, and then on examining with a power of 135 diameters one of the smaller spherules at its margin, where the envelope of leucocytes is a little detached, the appearances presented in Fig. 2 are seen, and are, I think, such as suffice for diagnostic purposes.

I do not propose to deal here with the histology of these colonies, but I may briefly point out that the envelope of leucocytes appears to be maintained in its position by a number of fine radiating filaments, some of them with small club-shaped terminations; and these radiating filaments are external to the more closely approximated and swollen clubs, which present the higher refractive index and usually the yellowish or greenish colour. When it is not convenient to employ the microscope thus upon fresh colonies, they may, if they have been preserved in spirit, be sufficiently well seen for diagnostic purposes if they be softened in moderately weak solution of potash, and then soaked and mounted in glycerine (see Fig. 3, which shows a colony thus prepared and magnified 75 diameters).

No relief followed this operation. The implicated regions remained swollen, painful, and tender, and about the iliac region and flank the skin became red, and an eruption of vesicles appeared, which soon became confluent and at length excoriated. By its distribution this eruption seemed to be due to the local irritation of the discharge. Slight diarrhœa came on for a few days, and the general condition became worse.

April 21st.—He was seen again by Dr. Pye-Smith, and on May 2nd by Sir James Paget. After this he took freely of morphia, was rather too somnolent, at times delirious, and on May 6th began to pass urine in bed.

May 8th.—Some increase of diarrhœa, but no actinomyces found in the stools.

12th.—The eruption appeared on the prepuce and scrotum.

21st.—Had a severe rigor, with rapid brief rise of temperature to 102·6° F., great prostration, pulse 156,

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respirations 50. Alcohol was freely given, and it seemed to help him to rally.

23rd.—A striking diminution had been noticed for a few days of the local œdema about the flank and of the granulations at the openings. After this he gradually lost flesh, strength, and intelligence, was very restless and distressed, but not in great pain. The temperature rose to a higher average, and on June 19th he had another rigor with a temperature of 103° F. He died on June 21st, having shown no evidence of any lung complication except an irritable cough for the last three days of his life.

Unless the rigors mentioned above were related to a metastasis there were no distinct indications of any such event having taken place throughout the course of the malady, and there were no such disorders of the functions as pointed to any viscus so affected. Palpation and percussion often made during the illness revealed only some enlargement of the liver without change of form or increase of solidity, so that its border was never distinctly felt. The abdomen remained rather large, especially in the hypochondriac regions throughout, certainly it was not reduced in the same proportion as the rest of the body. The vesicular eruption mentioned above appears to have been infective, as it appeared once or twice on his face after scratching. During the short period, some fortnight before death, in which the local swelling was so much reduced, deep palpation was again possible, and a deep-seated solid resistance was felt in the iliac region like that described on January 4th, but much smaller.

The venous turgescence and œdema of the right lower extremity which appeared on December 5th, 1888, as mentioned above, had almost disappeared in about a month, but the more localised œdema which returned about January 10th, 1889, persisted in some degree to the end, affecting the scrotum about the middle, and the left leg towards the end of March, 1889. Gradually, although the œdematous skin remained pale and the

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smallest venules and capillaries were not distended, the larger veins on both sides of the abdomen became too visible, and in June, 1889, the superficial epigastrics on each side were visibly continuous with the thoracic veins. It was thought that the venous thrombosis had extended to the ascending vena cava. Throughout the illness the urine was abundant, free from albumen and from sugar, normal in aspect, and contained, on the single occasion on which the examination was made, 1·5 per cent. of urea.

The heart was examined each time before an anæsthetic was given, and was found normal. There was at the end great wasting of muscle and of fat, notwithstanding the large quantities of food taken, his inactive existence, and his apparently fair digestion.

It is noteworthy that of the numerous congenital lipomata met with under his skin, some had, it was believed, quite disappeared, and nearly all had diminished in size. The colonies or spherules of actinomyces were still met with in the discharges up to the last, but their numbers were much reduced. At different times during their appearance some of the spherules were sent to three different pathological laboratories in London, and also to Cambridge, for artificial cultivation, but these attempts did not succeed.

Autopsy (June 22nd, twenty-five hours after death).—Very little decomposition present, slight rigor mortis. The œdema very perceptible in both legs, more especially in the left. The blood had, however, disappeared from the surface, and except in the dependent parts the skin was very pale. The capillary engorgement which during life had been so marked a feature of the right iliac region and flank had disappeared, and in this region the swelling was also very much reduced. The superficial epigastric veins were no longer striking objects, nor was their continuity with the thoracic veins at all noticeable. There was, however, a little brown stain in the skin where it had been so long inflamed, and where it had been excoriated there was seen a dry brownish scale. The openings

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of the sinuses gaped, and were almost dry; the whole region was much reduced in palpable as well as visible swelling, having become bloodless.

Thorax.—Very little obvious change on first opening the body. The lungs pale, especially in front, with marked black marbling. Left apex adherent, slightly puckered, on section pale in front congested behind, and œdematous. Right apex more firmly adherent, and puckered and firmer, and more congestion seen on section. No solid masses seen anywhere in either lung; a few small scattered recent patches of lymph on pleura. No adhesion of lungs to diaphragm.

Heart normal in size, colour, and texture of muscle. The mitral, tricuspid, and pulmonary apertures and valves normal. The aortic orifice normal, the valves competent, but there were small hard nodules upon two of them. The ascending aorta was somewhat dilated, and there were several patches of atheroma upon its surface, one of which was eroded. Only the usual post-mortem clot seen in the right heart.

Abdomen, when first opened, presented no strikingly abnormal aspect, except that the edge of the liver was far too low, although its surface was smooth and yielding to the touch. There were no striking indications of recent peritonitis or of enteritis. The intestines were pale with rather scanty contents, stomach empty and collapsed. Near the cæcum a few loose films of recent lymph were seen, and deeper in the pelvis a small amount of clear serum.

Liver was removed, and seen in so doing to be enormously large, without much change in form except a slight diffuse prominence at the most convex part of the right lobe, where there was a patch of dark slate colour like a post-mortem stain. This part was also thicker than normal, and suggested the presence of a deep cyst. The organ as a whole was soft and greasy to the touch, and on section showed a coarse mottling with portal venous congestion in patches. Section through the pro-

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minent part of the right lobe showed there, close under the surface, without, however, involving its tissue, a metastatic focus fully as large as the doubled fist, having a somewhat spherical form and soft consistence. In the centre it consisted of a pale greyish sort of muco-pus, only more ropy and consistent than is usually seen; it scarcely flowed at all on section, and was contained in a thick soft tissue of altered liver without any definite wall or limit. It was not possible to make even fairly good sections, so some roughly cubical masses of the outer portions were cut and put into methylated spirit for examination, and some of the softer central mass separately preserved. There were no other foci in the organ, and nowhere was the capsule of the liver or the peritoneum inflamed.

Spleen large, about twice the normal size; no deposits in it.

Kidneys large, rather coarse in structure to the naked eye. The left rather soft to the touch, without visible deposit. The right rather firmer and similar in aspect, but with a few minute pale points which might be deposits. A sample was placed in methylated spirit.

The whole length of the intestinal canal appeared normal except the cæcum and the parts immediately adjacent; here there was slaty-black discoloration visible behind the outer border from the vermiform appendix upwards as high as the kidney, and there was thickening of the peritoneum with some old adhesions so mixed up with the thickening as not to be separable. The vermiform appendix was tortuous, bound down, and almost lost to sight; touch was more helpful than sight here, and by it marked induration was felt behind the cæcum, and both above and below it. This induration was certainly much less, at least outwards in the direction of the flank, than observation during life had led me to expect. It extended inwards so as to be continuous with the tissues upon the right side of the bodies of the vertebræ, was traceable downwards along the great vessels to Poupart's

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ligament, and a short distance over the margin of the true pelvis in the direction of the rectum. Everywhere, however, the region was free from either purulent accumulation or solid masses; the long drainage had emptied old collections, and no large solid masses probably had ever existed.

The ascending colon was divided and removed together with a short piece of the ileum; and in doing so one had to divide the indurated walls of a considerable cavity behind the cæcum, which extended from the sides of the bodies of the vertebræ outwards to the sinuses, upwards nearly to the kidney, and downwards along the great vessels nearly to Poupart's ligament; this cavity was very irregular in form with anfractuous walls, a very maze of communicating channels, for the most part with thin soft walls in front, and nowhere either very hard or thick. The firmest parts of the walls and the thickest were towards the psoas muscle, and about the neighbourhood of the vermiform appendix. In removing the cæcum with the vermiform appendix a rather wide sweep was made with the knife through the thickened tissues, and here portions of the common iliac vein and artery with a short length of the external iliac vessels were in part removed with the gut, just where they were involved in the thicker parts of the abscess wall. After removal of the gut the posterior walls of the abscess were exposed, lying in and on the psoas magnus and neighbouring muscles, and in close proximity to the ascending vena cava, nearly as high as the right renal vein. The ascending vena cava was full of clot, soft, deep red, and easily falling out in the upper part near the right renal vein; firmer, whiter, and attached to the walls lower down near the common iliacs, which, as well as all the veins below, seemed to be blocked. The vena cava, together with the left common iliac vein and its branches down to Poupart's ligament, was removed without difficulty; but the right common iliac vein and its affluents were so involved in the indurated walls of the abscess behind the cæcum that they

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had to be separately removed, part being engaged in the wall of the abscess removed with the cæcum, and part subsequently being cut out from the walls left behind. The removed cæcum and appendix on being opened showed a fairly normal aspect of the mucous membrane, and no ulcerated aperture was found. There was no mycelium seen, such as has been described by Chiari. Near the aperture leading into the vermiform appendix the mucous lining was thrown into strong transverse and concentric rugæ, corresponding to a considerable induration of the tissues behind the gut. The vermiform appendix was bound down, covered and obscured by adhesions and thickening of the peritoneum, which were continuous with that part of the abscess wall. The aperture of entrance from the gut into the appendix was patent.

On further examination of the removed cæcum and appendix it was found that the vermiform appendix was dilated immediately beyond the aperture from the gut, that this dilated part had irregular walls, and that it had more than one communication with the abscess cavity. No continuous channel could be discovered between this dilated part and the rest of the vermiform appendix, which was turned here at a right angle, and was apparently closed at both ends. There was no discoverable enlargement of the mesenteric glands. One mass like a large *lymphatic gland* was removed from the right groin, as it had been noticed during life; but on inspection and section it appeared to be a firm lipoma, which had been somewhat inflamed.

The other viscera were not examined. The walls of the abscess and sinuses showed, when closely inspected, even with a lens, no indications of any actinomyces spherules *in situ*, and on careful search among the débris washed from the specimen I found only a very few of them.

The spherules which had passed in the discharge, as well as those found after death and preserved in spirit,

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were examined in various ways, so that I satisfied myself of their being truly colonies of actinomyces; and in similar ways I examined the spherules which were found in the soft semi-fluid centre of the hepatic focus, with a like result.

In these examinations, made by teasing, &c., without sections or staining, I relied chiefly upon the radially arranged threads, tending to form groups which gave to the whole colony a sort of morula aspect, upon the evident branching of these threads as they advanced to the periphery, and upon their tendency to present a club-shaped form when they could be isolated either at the periphery or when broken off. I think the tendency of the elaborate researches on actinomyces by Boström (see Ziegler's 'Beiträge,' Bd. ix) is to reduce the diagnostic value of the thick, firm refractive clubs.

Afterwards sections were made by my son, Dr. W. B. Ransom, by the paraffin method, and stained by the Gram process (gentian violet and eosin), of the walls of the metastatic focus in the liver, which showed remarkably well the colonies of actinomyces *in situ*. Similar sections were made by him also of the walls of the abscess behind the cæcum and of the vermiform appendix, where it was dilated, eroded, and communicated with the abscess; but in these no such colonies were found *in situ*. Neither were they met with in similar sections of the external iliac vein just where it was almost included in the indurated wall of the abscess. The separate spherules, which had been at various times discharged from the abscess when similarly embedded and cut in paraffin, exhibited the characteristic and well-known structure. In sections, however, by this process the eosin did not bring out the separate clubs. The fragments of soft tissues, the walls of the sinuses which had been squeezed out and preserved in spirit after the operations of February 28th and April 13th, were also similarly cut and stained and examined for actinomyces colonies without result. They consisted apparently of granulation tissue, much of it modified by

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the thrombosis and œdema, and exceptionally vascular, so that it is not surprising that at the earlier examination of these fragments, made soon after the operation of February 28th, no evidence of the true nature of the malady was furnished. The only thrombosed vein thus examined was the right external iliac near to the common iliac. It was here reduced to a mass of scar tissue and organised clot, with numerous canals in it. The other veins were not thus examined, but judged of by their aspect to the naked eye they did not present the appearance of actinomycosis. I concluded that the coagulated mass in the veins was not specifically affected by the ray fungus.

Search was made for bacteria by the methods of Loeffler and of Gram in the sections of the liver and of the granulation tissues of the sinuses removed by operation without result, but in sections of the abscess wall through the eroded vermiform appendix numerous thick bacilli were seen well stained by the method of Gram. There can be no doubt, however, but that numerous bacteria existed in the pus of such sinuses.

Remarks.—On reviewing the case, I think it may be said that the first diagnosis was in part justified, and in part not. I had considered the clinical features conclusive as to the existence of a perityphlitis, and when in the progress of the case evidences of a fæcal abscess appeared this view was strengthened. I had also adopted the view that the vermiform appendix was primarily diseased, and that such disease was due to a fæcal concretion of some kind. In this respect the event showed that I had fallen into an error, which was not removed until the spherules of actinomyces were found in the discharge.

In the main my colleagues were in accord with me in their opinions, being, however, more cautious than I had been, and not responsible in any manner for that part of the diagnosis which events proved to have been wrong. There was a period during the case, especially when the exploring needle failed to reach pus, and before

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a distinct point appeared, when the question of a growth was considered either as an alternative diagnosis, or as a supplemental one. Mr. Symonds was, I think, rather inclined to sustain the diagnosis of a growth of some kind, either sarcomatous or carcinomatous, but by the medical attendants as a whole these views were held in suspense.

Had I made, as I ought to have done, an adequate examination of the earliest discharges on February 16th, I cannot doubt but that the diagnosis might have been made two months earlier than was the case. The rule carefully to examine all discharges is one I have long accepted if not always followed, but the importance of it is well seen in such a case as this.

The examination of sections of the fragments of tissue obtained during the operation was made as soon as opportunity served by Mr. Symonds and by myself, but gave no satisfactory help in the diagnosis. It only showed what in other ways was learned, that an inflammatory process was in operation, and both destructive and reparative processes were going on side by side. Afterwards, as a result of the examination of many sections of the abscess walls, of the hepatic focus, of the peripheral inflamed tissues in the neck and face of another case, confirmed by reading the reports of various observers, I felt less surprised that these sections gave no ready clue to the nature of the case. For it appears to be the rule in man that the ray fungus produces an inflammation which is suppurative in character in its immediate vicinity, and protective through a peripheral zone of rather considerable thickness; so that except in comparatively rare cases, and only then if the tissues have been carefully embedded in either celloidin or in paraffin, the sections would not show the ray fungus *in situ*, even when properly stained. In short, from the readiness with which the spherules fall out and pass away in the discharges, it is a much more facile way of diagnosing a case to search the pus than to examine the tissue.

It is a question of much interest from a diagnostic

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standpoint to decide whether or not the general clinical characters of the malady suffice to distinguish it, or at any rate to raise a suspicion as to its nature, such as might direct the inquirer aright. I think the answer must be that neither the local nor the general reactions are sufficiently characteristic to permit of a positive diagnosis, but that the local reactions when observed through a sufficient period of time, or when a good history of their previous course can be obtained, suffice for a provisional diagnosis such as may guide the observer.

The case recorded by Boström (p. 168, Ziegler's 'Beiträge,' Bd. ix), in which a tubercular swelling under the jaw was diagnosed as actinomycosis, shows that even in the hands of experts a certain diagnosis is at present not to be made without a microscopical examination of the swelling or its contents. That a probable one is possible, is evident from two other cases reported by the same writer (l. c., pp. 13 and 21), in which the diagnosis was made before any opening appeared, and the whole mass on removal and examination proved its correctness. That the external examination of actinomycotic swellings may suggest a sarcomatous growth is shown in the case of this disease reported by the same author (l. c., p. 29), in which the operation was undertaken on the supposition that the swelling was a malignant sarcoma.

These difficulties of diagnosis become still more marked in cases like the one here reported, where a hollow viscus is involved, and various other pathogenic organisms and substances become factors. The case before the Society had also a further and, as I think, an accidental complication tending to obscure the regular course of an actinomycosis, viz. the thrombosis of the great veins and the consequent venous turgescence and œdema which began early in the case, and never ceased to affect its aspect in some degree until the end.

However, I think from my observation of this case, and of two others which I have since had the opportunity of seeing, as well as from such reports as I have read,

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that when a subacute or chronic inflammation occurs which spreads without much elective affinity for any tissue or viscus, but which tends to spread in all, although in the main downwards, which is hard, even woody to the touch, involves the skin, and softens in the centre slowly, at length yields pus in rather scanty proportions relatively to the bulk of the swelling, and does not heal when freely opened and drained, it is almost certainly an infective malady, and actinomycosis may be strongly suspected, and the spherules or colonies should be sought for.

It may be perhaps assumed that in this case the earliest seat of the disease was in the vermiform appendix, that here a fragment of or a whole grain of corn or of grass lodged, and the actinomyces parasitic upon it developed, and the destructive inflammatory processes it set up in its immediate vicinity perforated the posterior walls of the appendix, so, however, that being all through accompanied by a peripheral protective process leading to adhesions and limitations, the contents of the appendix escaped only into the connective tissue behind the cæcum; then doubtless the factors at work in exciting the further inflammatory process were more complex, as the intestinal contents are known to be very varied, and many of them pathogenic. Even the peripheral or protective inflammatory process appears, however, in this case as in many others, to have been in large part responsible for some of the most striking and painful of the symptoms.

Without taking up too much time by trying to describe all these effects, I may note that the venous thrombus, with all its grave and striking results, was a direct consequence of the vein having been involved in the peripheral zone of the inflammation.

Looking back, the question is a proper one to ask, could we, by an earlier clear diagnosis, or in any other way, have found good grounds for a different and successful treatment? I fear not. Although great success has attended the surgical treatment of actinomycosis of super-

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ficial parts, as yet I know of no satisfactory mode of treating visceral actinomycosis.

While I was under the domination of the idea that the disease was essentially an ulceration of the vermiform appendix due to a fæcal concretion, I had some hopes that Mr. Symonds might be able to find such concretion, remove it and the vermiform appendix, and possibly even at the cost of some risk to life bring about an imperfect recovery. Mr. Symonds, however, during the operations which he performed, made it clearly manifest that such a course was not practicable or justifiable; and further, that he could not ascertain the existence of any growth or mass which could be removed so as to benefit the patient, and with this conclusion all the medical men in attendance agreed. It may be a question, however, whether or not in this case the bold and adventurous course which has been recommended, of removing the vermiform appendix quite early in the illness, might not have had a favorable termination. On the whole I think that the infective character of the malady would have led to a disastrous result, and that the conclusion which we all came to at first not to advise such an operation was justified, seeing that this was a first attack.

If any question of operation arose in a case of visceral actinomycosis, it would be of great value to know whether at the time any other important part was affected, especially if any metastasis had occurred. This case unfortunately does not at all tell us when the metastatic focus in the liver began. Considering its large size, it could hardly have been so late as the first rigor on May 21st, just a month before death. There were no sufficiently definite indications locally ascertained during life, and it may be said that although on pathological grounds the hepatic focus was expected and sought for at the autopsy, it had not been diagnosed during life. The fever, which persisted in some degree with few and short cessations for the last seven months of the eight during which the illness endured, was, I conclude, neither directly a result of

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actinomycosis, nor of the metastasis, but in all likelihood due to the action of other pathogenic factors derived from the intestinal canal. There can, I think, be little doubt that the metastasis took place through the portal vein, and the absence of any such foci in the lungs strengthens the opinion formed on other grounds, that the thrombus in the vena cava and its branches was not affected by the ray fungus.

Although up to the present, treatment has failed in visceral actinomycosis, it is conceivable that a successful result might follow in certain cases where the primary focus is within a moderate distance of a depending opening to the surface, either natural or artificial. For, apart from the statements made about the occasional cure of this disease in cattle after free discharges, there is some ground for adopting such a hopeful view, founded on the facts observed in this as well as other cases recorded, viz. that the exciting cause of the destructive inflammation is thrown off freely, and that in long-standing sinuses but few spherules are left behind.

These hopes are strengthened also by the tendency so generally met with for the periphery of the disease to form a defensive scar tissue. There is also some ray of hope to be found in the chance that injections of not too poisonous or escharotic substances might in certain positions reach the extreme limits of the sinuses, and there restrain the further growth of the ray fungus.

I ought, before closing these remarks, to call attention to the facts noticed by Boström and others, that in some cases the spherules of actinomyces are translucent and colourless, and then easily escape observation; in others, although more or less opaque, they have only a pale grey colour. In each case my description of them would prove insufficient. That that description, however, holds good for a majority of the cases I do not doubt, judging from the testimony of various observers, and from the three cases I have had the opportunity of seeing.

The case here recorded was the first I had seen, and

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I was not familiar with the appearance of the spherules, nor would they have attracted my attention probably had I not been searching in the discharges for evidence as to the relation of the abscess with the gut.

I availed myself, therefore, of the writings of various authors, but more especially I have to express my obligations to those of J. Israel, Partsch, Crookshank, and Boström, the last of whom has made the most exhaustive and recent researches in actinomycosis with which I am acquainted.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 16.)

DESCRIPTION OF PLATE I.

Actinomycosis of the Vermiform Appendix causing Perityphlitis (Dr. W. H. RANSOM).

FIG. 1.—Spherule or colony of actinomyces, $\frac{1}{15}$ inch in diameter, from discharge, seen in water as transparent object with lens. Magnified 10 diameters.

FIG. 2.—Marginal part of a smaller spherule, showing the peripheral radiating and branched threads and some small clubs. Magnified 135 diameters.

FIG. 3.—Smaller colony, $\frac{1}{35}$ inch in diameter, from spirit, treated with solution of potash and mounted in glycerine. It shows the rays very delicately with oblique light. Magnified 75 diameters.

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x 10

Fig. 1

x 735

Fig. 2.

x 25

Fig. 3.

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A CASE

OF

ACTINOMYCOSIS HOMINIS OF THE URINARY AND ALIMENTARY TRACTS.

RECOVERY.

BY

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Received April 13th—Read November 10th, 1891.

W. K—, a married man, æt. 48, came to me on January 29th, 1891, complaining of "lead-poisoning." He said he had been quite well up to September, 1890, when a few days after painting his kitchen he was taken suddenly ill with colicky pains and vomiting. This attack subsided in a day or two, but since then he has been liable to indigestion, loss of appetite, occasional colic and vomiting, distension of the stomach, wind and constipation alternating with diarrhœa. He has not noticed anything peculiar in regard to his stools.

The urine has been thick at times; he has sometimes got up thrice in the night to pass water, and has occasionally had a little scalding pain at the end of the penis.

He has lost two stones in weight in the last six months.

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His wife thinks he began to lose flesh and general vigour about the end of July, 1890.

Past history.—The patient has lived all his life in the north of Notts. When a young man he worked for three years on a farm, where he milked the cows. He then worked for a few years on a railway, but for the last fifteen has been settled as an insurance agent in Mansfield, and has had nothing to do with cattle.

Until now he has had no serious illness, but while employed on the farm he had a sore on the heel, which discharged for six months and then healed perfectly.

He has frequently in the summer chewed fresh ears of corn—wheat, barley, and oats,—and remembers last June eating both wheat and barley. He has not been in the habit of eating raw meat or vegetables.

State on January 29th, 1891.—On examination on January 29th the patient showed no signs of lead poisoning. He was pale and rather worn-looking, but not excessively emaciated. Heart and lungs normal. The abdomen was considerably and uniformly distended, and tympanitic on percussion. Loud gurglings were occasionally heard, and movements of the intestines were visible. The solid viscera could not be felt. Examination of the rectum showed a little tenderness of the prostate, which was, however, of normal size and shape. Above the prostate was felt an irregular firm swelling, apparently placed in or just in front of the anterior wall of the rectum.

Some urine passed in my presence was acid, and high-coloured, and deposited a quantity of small oval, translucent, jelly-like flakes of a pinkish colour, and about half a dozen granules, which placed under the microscope showed distinctly the characters of spherules of actinomyces. There was also a very little pus, and a small quantity of albumen.

The translucent "flakes" were mostly flat ovals, finely fibrillated in concentric rings, and turned opaque with water, but did not turn opaque or shrink with acetic acid. On one of them was a small prostatic concretion, and a

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few spermatozoa were also found among them. The urine contained no casts, blood, sugar, bile, or fæcal products, and no bubbles of gas have been noticed during micturition.

Progress of case.—During the next ten days numerous specimens of urine were examined, and in all were the Actinomyces spherules found, *e. g.* from six to twelve in a six-ounce bottle of urine. The peculiar flakes I have not found again.

On February 2nd the patient brought me a portion of a slimy stool, in which were a few streaks of blood and two or three spherules of Actinomyces.

On February 11th he was admitted under my care into the General Hospital, Nottingham. The note made on February 15th remarks :—He has little to complain of but wind and distension of the abdomen. In especial, he makes no complaint about his water. The skin is everywhere healthy, the old wound on the heel being perfectly healed. His respiratory, circulatory, and nervous systems present no sign of disease. The temperature is normal. The appetite is fair, the bowels rather constipated; there is much flatulence, but no nausea or vomiting. The teeth are mostly decayed, and many are absent. Gums rather retracted and spongy, and bleed readily. There is no sign of alveolar abscess or of disease of the jaw. The pharynx and tonsils appear healthy.

In the abdomen the distension is somewhat less, but there is no other change. The stools occasionally have a little slime in them. The lump felt by rectal examination above the prostate is tender, fixed, firm, and feels about the size of an orange. The posterior wall of the rectum is quite moveable. There is slight frequency of micturition—once or twice in the night,—but no pain or vesical tenesmus.

Urine, about thirty ounces per diem, is neutral or faintly acid; sp. gr. 1020—1026; it is sometimes quite clear, sometimes turbid with urates, a little mucus and a very little pus. There is sometimes no albumen, some-

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times a trace. Urea, 2 per cent. Most specimens contain from one to a dozen spherules of actinomyces. No other abnormal elements. The spherules are of a creamy or yellowish colour, no brown ones being found.

The scrotum, testes, and penis are normal.

The patient was ordered terebene by the mouth, with a mild aperient pill, and for a few nights had enemata with turpentine, with the result that the abdominal distension was considerably reduced, although it still tended to recur.

During the next ten days the bowels were opened regularly by the enema or pill; and the fæces, though unformed, became in other respects normal. The slime and blood disappeared, and I have never, except on the occasion already mentioned (February 2nd), found the fungus in the stools. The spherules in the urine also decreased in number, and on some days none were found. The albumen and pus disappeared and remained absent after February 19th.

On February 27th occurred the first and only rise of temperature. There was general malaise, some griping pain in the abdomen, and the temperature rose to 102° F. There was no rigor. The next day the patient had seven loose stools and vomited once. No pus or blood was noticed in the stools. The temperature remained at 100° on March 1st, but returned to normal on the next day.

On March 2nd the lump above the prostate was thought to be rather smaller than before.

On consideration of the case it appeared clear that there was an actinomycotic growth communicating with the bowel on the one hand, and the urinary tract on the other, and I considered it probable that the mass felt above the prostate was the growth, which communicated by different sinuses with the rectum and bladder. It also appeared probable that the opening into the rectum was closing, if not already closed up.

That this mass was the seat of the disease was also the opinion of those of my colleagues who examined the case.

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After consultation it was accordingly decided to open the abdomen with a view to removing as far as possible the growth. It was felt that such a growth if left must prove fatal, and that, although complete removal might prove impracticable, it was right to give the patient the chance.

On March 3rd, therefore, my colleague, Dr. Herbert Owen Taylor, opened the abdomen by an incision from umbilicus to pubes. The result was somewhat of a surprise. There was no tumour, no abscess cavity, no large inflammatory growth, and no recent lymph. All that was found was a number of rather old vascularised adhesions between some coils of small intestine occupying the pelvis. Many of these were broken down by the operator, and a sufficient view obtained. Bimanual examination, with one finger in the rectum, could detect neither thickening nor ulceration of the wall of that viscus, and examination of the bladder, into which a sound was passed, likewise failed to show any morbid change. It was obvious that the mass which had been mistaken for the actinomycotic growth was really a piece of small intestine, containing fæces, which lay transversely across the pelvis, to which it was fixed by old adhesions.

No sign of the parasite was discovered within the abdominal cavity, though it must be added that to avoid further shock the upper part was not subjected to a prolonged examination.

The operation was therefore brought to a close, and the wound stitched up without washing out the peritoneal cavity, and without inserting a drainage-tube. The patient made an excellent recovery from the operation, had no rise of temperature and no pain, and on March 13th he was re-transferred to my care.

I may add that the bowels were opened freely by an enema on the fourth day after the operation, and that the stool, which I examined carefully, showed no sign of spherules, mucus, or blood.

During March, although the wound healed perfectly, the patient's abdominal troubles did not diminish. The

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distension of the intestines remained considerable, there were irregular attacks of diarrhœa, occasional vomiting, and anorexia, and he lost nine pounds in weight. The stools, though loose, did not show blood or mucus, nor were any spherules of the fungus discovered in them. The temperature remained normal.

On April 1st he was ordered β-naphthol in three-grain doses every four hours, and three grains of calomel on alternate days. From that date improvement has been unimpeded.

The distension of the abdomen disappeared, the appetite became good, he gained a stone in weight during April, and passed without discomfort one or two formed and apparently normal stools daily. The calomel was stopped after three weeks, but he continued to take the β-naphthol till the end of June, though all abdominal symptoms had ceased by the end of April.

The urine, during the period of intestinal trouble in March, became scanty, but, except for the presence of a few Actinomyces spherules and an occasional deposit of urates, was normal. The administration of Ol. Copaibæ for a few weeks raised the daily average from twenty to fifty-five ounces, and since then, up to the present time (October, 1891), he has passed about fifty ounces daily. There has been no undue frequency of micturition, and no pain during the act; but he has, throughout his illness, occasionally had "springing" feels at the end of the penis.

During April and May a soft catheter was several times passed into the bladder, and the urine thus drawn off has never contained any spherules, except on one occasion when the instrument was withdrawn while the last portion of fluid was escaping, so that a spherule might easily have entered the eye of the catheter during its passage along the urethra. The bladder has also been washed out with boracic acid lotion without bringing away any spherules, although they still recurred in the urine as passed.

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The patient was sent home on May 9th, and has since felt in perfect health. He has gained two stones in weight, has no digestive troubles, and can walk twenty miles a day. Symptoms of disease of the urinary tract have been absent except for occasional slight "springing" sensations, felt chiefly at night, at the urinary meatus. The urine, which has been otherwise normal, continued until the second week in October to show a few spherules of Actinomyces, but since that date *none have been observed.* While at home the patient measured his urine, collected the daily sediment in a bottle which I weekly examined, and on October 20th he again came into hospital for closer observation. There is no sign of extension of the disease into the abdomen; the prostate is of normal consistence, not tender, and I think a little smaller than it was, and no sign of disease is to be found in the penile urethra. The "springing" sensations are diminishing, and the patient sleeps all night without needing to pass water (October 30th).

Remarks.

Diagnostic.—The diagnosis of actinomycosis, though most unexpected, presented no difficulty when the patient passed water. The yellowish spherules, about half the size of a pin's head, were to the naked eye suggestive, and examination under a low power of the microscope of a fresh spherule in a drop of urine and slightly pressed by the cover-glass at once showed the characteristic yellowish-brown mulberry mass of the Actinomyces colony, with the usual outer layer of pus-cells. Even before the use of a high power there was no doubt of the diagnosis. Some points concerning the structure of the fungus I shall refer to later, but will now consider the probable seat of the disease.

The patient's troubles for the first seven months of his illness were chiefly connected with the alimentary canal. Intestinal irritation and intestinal atony were the prominent features of the case; and the discovery on one

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occasion (February 2nd) of a few spherules in the fæces shows that the parasite was actually in the intestinal canal. The intestinal troubles, however, though for long persistent, have vanished. There is now neither symptom nor sign of disease of the alimentary canal, and no spherules have for eight months been found in the fæces.

In the urine, on the other hand, the spherules, though less abundant, continued till lately to occur, but caused practically no symptoms.

The absence of fever, of general peritonitis, of evidence of deposits in the liver or lungs, make it very improbable that metastasis has occurred, and that there is more than one focus of disease; so that we have to consider in what place has the actinomycotic growth spread from the alimentary to the urinary tract. (A spread in the reverse direction is extremely improbable.) It is a well-known characteristic of Actinomyces to grow without respect to boundaries from one organ to another, and it is conceivable that either kidney might be invaded from the colon, or the bladder or ureters from the small intestine or rectum. The absence of renal symptoms, and the fact of the urine being normal in every respect except in containing the spherules, render it improbable that the kidneys are affected, and the operation showed no sign of any inflammatory growth invading the bladder or ureters. Moreover the facts that bladder symptoms are absent, and that the urine drawn off by a catheter almost invariably contains no spherules, contra-indicate the view that the bladder is the seat of the disease.

The view to which I incline is that the prostate gland has contained a small actinomycotic focus, and that invasion occurred from the rectum. I imagine that the fungus located itself in the front wall of the rectum, and thence spread forwards till it burst into the prostatic urethra, but that, as not unfrequently happens in this disease, the rectal sinus healed while the urethral continued to discharge. The occasional presence of prostatic calculi among the spherules supports this view.

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Against this view may be urged the absence of a considerable enlargement of the prostate or marked alteration in the consistency of the gland, there being neither fluctuation nor the wooden hardness sometimes found in actinomycotic growths. But it is very possible that the free drainage which has gone on will account for this peculiarity.

But, even if the rectal ulceration be healed, we have still to ask ourselves the question whether there may not still remain a deposit of the parasite higher up in the intestine. A remarkable case has been reported by Chiari, of Prague, in which the large intestine of a man who had died of progressive paralysis was found to contain on nearly the whole of its mucous membrane a diffuse film of actinomyces, which nowhere caused deep ulceration, and which was not known to have caused symptoms during life. It would seem that in the intestinal as in the respiratory tract the fungus may present two different habits; one a superficial, not destructive growth, the other parenchymatous and destructive. It is impossible to say positively that an actinomycotic deposit of the former character is not still present in my patient, though there is little probability of the existence of the parenchymatous form. And the marked recession of all symptoms of intestinal disease, with the gain in weight and general vigour, which has been maintained since the commencement of the β-naphthol treatment, leads to the hope that the fungus has been completely removed from the digestive tract.

Prognosis and treatment.—Although partial healing of an actinomycotic abscess may occur, and a tendency to encapsulation has been observed rarely in animals, I am not aware that any case of cure without operative interference by knife or caustic has been reported in man.

In the present case it seems probable that the disease is no longer present in the intestine; and it is possible, though we cannot as yet speak with certainty, that it is disappearing, or perhaps even gone from the urinary tract.

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The free drainage that has been maintained into the urethra seems to have prevented growth inwards, and the fact that prostate and bladder still show no sign of disease, although the discharge has ceased, justifies the hope that the tissues have gained the victory and encapsuled or expelled the parasite. Some months must, however, elapse before a confidently good prognosis can be given.

With regard to treatment, the facts related offer, I think, fair grounds for concluding that the disappearance of signs of the intestinal disease was largely due to the enemata of turpentine at first, and the course of calomel and β-naphthol afterwards. After the stoppage of these drugs at the end of June the patient began to take Liq. Hydrargyri Perchloridi with iodide of potassium, and since September 24th he has taken the iodide only in doses of a drachm daily. During this time he developed an iodide rash on the face, and for the first few weeks no marked effect on the number of spherules in the urine was observed. The absence of spherules, however, since October 15th shows that the drug has done no harm, and may possibly have contributed to the suppression of the disease.

P.S. (April 5th, 1892).—The patient has remained free from all signs or symptoms of disease since October 15th, 1891, and to-day showed himself apparently in perfect health. No spherules have been found in the urine, although sought for. A sample examined to-day was normal. He has taken no medicine since October.

The lapse of six months without any return of the disease affords considerable support to the conclusion that the recovery is complete.

(August 7th, 1892.)—The patient is still in perfect health.

Ætiological.—The present case is of some interest in regard to the ætiology of Actinomyces. There is absolutely no evidence of it having been caught from cattle. (The sore heel twenty-four years ago can hardly be connected with the present illness.) There is, on the other

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hand, evidence throwing suspicion on what Boström and Johne and others have accused, viz. ears of corn, and especially barley. The patient can recollect picking ears of barley at the end of June, and about the end of July his health seems to have begun to fail, though serious symptoms did not develop till September.

In connection with the remarkable fact of three cases of actinomycosis hominis having occurred in the last two years in the neighbourhood of Nottingham, I have made some inquiries of veterinary surgeons as to its occurrence in cattle. There seems no doubt that by many the disease is not recognised, and information has proved scanty.

But Mr. Brett, M.R.C.V.S.,[1] who for six years until lately practised as a veterinary surgeon in Mansfield, told me that he frequently saw cases in that neighbourhood. In one case it broke out in some calves, who, contrary to the usual practice in that district, had been littered on barley straw. Mr. Brett advised the substitution of oat straw, and this was done. But although the cow-shed had been disinfected, the next lot of calves got the disease even worse than the first; and Mr. Brett then found that although the barley had been taken out of the litter it had been put into the fodder. After complete exclusion of barley from the shed no more cases occurred.

I understand that in Norfolk and Lincolnshire, where the disease seems most common, barley straw is largely used for litter.

During the month of September I have examined a large number of specimens of barley, both ears and stalks, from the Mansfield district, but have found nothing that could be identified with the ray fungus. Smut is almost universal, and I also observed a few Leptothrix filaments containing spores. These filaments closely resembled threads of Actinomyces, as seen in a cover-glass preparation; but as no branching ones were seen, and no tendency to aggregate into colonies, there was no evidence to show a relation.

[1] I regret to say that he succumbed this summer to an attack of influenza.

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But on this point the experiments of Liebman[1] are of great interest. This observer planted beans, wheat, and barley in earth which had already been sown with a pure cultivation of Actinomyces, and found on them after the plants had grown, to all appearance normally, the rods which appear to be characteristic of certain stages of the life of the ray fungus.

It seems probable that the Actinomyces, if found on or in cereal and other plants, will not appear as spherules, but in the form of rods and threads, such as it often takes in artificial cultivations.[2]

Morphological.—I have been able to examine the structure of Actinomyces in specimens from the urine of my own case, both fresh and preserved, and in sections of the hardened tissues from the cases of my father and Mr. Anderson.[3]

Observers differ chiefly in their views as to the nature of what has been considered the chief characteristic of the ray fungus, the clubs; and to these, therefore, I will first refer.

Two facts help to explain the discrepancies of authors. The first is one mentioned by Boström and supported by my observations, that in young spherules the clubs may not be developed; and the second is that they are readily destroyed or altered by various reagents.

In bovine Actinomyces, where the clubs are so conspicuous, certain chemical changes appear to be undergone by the fungus sooner than in the human form; thus calcification is more common, and the clubs become early resistent to reagents.

The specimens obtained from the urine of my patient were probably all fairly young; they were all small in size, and all whitish or pale yellow in colour. In some of the smaller ones, examined directly after micturition, no trace of clubs could be discovered. And in those

[1] 'Arch. per le scienze med.,' vol. xiv, No. 18.

[2] Boström, 'Ziegler's Beiträge,' Bd. ix, 1890; Israel and Wolff, 'Virchow's Archiv,' Bd. cxxvi, Heft 1, 1891.

[3] Reported in this volume, pp. 63 and 103.

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spherules which bore clubs abundantly these structures were not, as the drawings of some authors suggest, at first sight the most conspicuous objects. The most obvious feature in many Actinomyces spherules are the radiating and sometimes branching filaments which project from the edge of the colony. Pl. II, fig. 1, shows a portion of a fresh spherule in which the clubs were as conspicuous as any I have seen. Even here the filaments are more numerous and project further than the clubs.

In deciding as to the existence of clubs it is necessary to be aware of their behaviour to reagents. Broadly I have found that acids preserve, while alkalies dissolve them. Fresh acid urine proved an excellent medium for their study, but so soon as phosphates were precipitated the clubs disappeared. Very weak potash was sufficient to dissolve them. Tap water caused most to disappear after a little time.

Salt solution of 10 per cent. shrivelled them, while 5 per cent. showed them excellently. Alcohol caused them to shrink considerably, and heat broke them up into fragments.

These facts may explain the frequent failure to demonstrate the clubs from human autopsies where post-mortem changes may have altered their characters.

The clubs, whether fresh or after treatment with alcohol, stained with eosin, and still better with rubin (Crookshank) (Pl. III, fig. 3).

The true significance of the clubs is one of the most important questions connected with Actinomyces. The view at one time held that they were comparable to the asci of lichens has lately lost ground, but Professor Crookshank, who has paid much attention to the subject, in his latest writings still compares them to the basidia of the higher fungi, and holds that they are organs of fructification.

Boström, on the other hand, in his recent exhaustive paper, brings forward strong arguments against this view, and considers them products of involution or degeneration.

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My observations agree closely with those of Boström. He describes the club as a gelatinous sheath on the ends of filaments, and finds no special tendency of the ensheathed filaments to form spores. The older the spherule the more conspicuous the clubs, and only in quite old artificial cultivations of the fungus does he find any clubs. He therefore concludes that the club formation is a sign of degeneration.

Of the existence of the axial thread in the club there is no doubt; Boström, Crookshank, and Delépine have figured it in specimens stained by Gram's method in combination with a red stain, and I have seen it both in such preparations, and in spherules examined fresh in urine or in 5 per cent. salt solution. This solution causes the filaments to break up largely into short rods and round coccus-like bodies, and brings out the clubs conspicuously (Pl. II, fig. 2). No cell-wall can be made out to the club, the hyaline substance of which shows frequently cross fractures, which in some cases are bridged by the axial thread.

The mode of fracture of the clubs, and especially the irregular shrinkings which they undergo on drying, are strongly suggestive of a jelly-like substance without cell-wall. The hyaline substance of the expanded club can sometimes be traced as a narrowing sheath down the filament, and in preparations stained by Gram's method with gentian-violet and rubin one can sometimes see on most of the filaments a narrow sheath of high refractive index with a faintly pink tinge. Moreover if a preparation showing clubs stained red with eosin be crushed, it will be found that nearly all the clubs have vanished, and that among the threads is an irregular mass of red homogeneous matter, which appears to be formed of confluent club substance. That the club-bearing threads are not specially differentiated organs is probable from the fact that every gradation in size from the smallest thread to the largest club is met with, as well as from the fact just mentioned that indications of a sheath of the same nature

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may be traced down the filaments. One may compare the club of Actinomyces to the capsule of the pneumococcus, which, in its behaviour to acids and alkalies, resembles it closely. Probably all the filaments of Actinomyces tend to secrete this mucin-like substance, which accumulates in larger quantity on the free ends of the older threads. In sections of a colony one often finds no distinct clubs, but a felt-work of threads, staining blue with aniline dyes, embedded in a homogeneous eosinophilous substance, in the periphery of which faint indications of radial striation may sometimes be found, while in others the periphery may be clearly composed of clubs. It would seem as if this homogeneous substance existed throughout the mycelium, the threads of which it held together (Pl. III, fig. 1).

There seems no evidence to connect the clubs with the reproductive function, but neither does it seem necessary to assume the opposite extreme—that they are products of degeneration. The secretion of a jelly-like substance is not uncommon in the Schizomycetes, and the regular occurrence of clubs in what are apparently healthy colonies appears to me opposed to Boström's doctrine on this point. It may be added that Bujwid, of Warsaw, has observed the formation of clubs in cultivations grown in an atmosphere of nitrogen.

Of the mode of reproduction of Actinomyces but little certain is known. At the margin of every fresh spherule one sees swarms of coccus-like bodies, and preparations teased and stained by Gram's method, or methyl-violet only, frequently show what appear to be spores in the course of the filaments (Pl. III, fig. 3). I have also seen clusters of these spore-like bodies adhering to a club, and rarely they are visible in the central filament of a club. But the readiness with which the filaments break up into rods like bacilli of various lengths, and into round bodies like cocci, suggests caution in forming conclusions as to the nature of these so-called spores.

Moreover in the sections of hardened spherules,

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whether from the urine or tissues, these "cocci" were not seen—a fact which suggests that they may be produced by the crushing or heating of a cover-glass preparation.

The study of Actinomyces in the tissues can only be satisfactorily made after embedding in paraffin or celloidin, as nearly all the colonies otherwise drop out. I have cut serial sections of the organs of my father's case, and of the parts removed by Mr. Anderson at the operation on his case, after embedding in paraffin, the sections being fixed to the slide by Mayer's albumen method and then stained.

It is remarkable, and somewhat annoying, to find what a large number of sections must be cut, even through the tissues which, to the naked eye, appear the focus of disease, before one comes across a colony. In other words, the tissues are changed for a long radius round the parasite.

The granulation tissue is remarkable and almost characteristic from the abundance of very large round granular cells undergoing fatty degeneration. Baumgarten, in his text-book on mycology, alludes to this, and I have certainly never seen anything like the granulation tissue from these two cases. There would seem to be a special tendency to fatty degeneration in this disease, for the liver in my father's case, far beyond the granulation tissue, showed the most intense fatty change.

As a rule, the colonies found by me in the tissues have been surrounded by a broad zone of leucocytes, many of which were degenerating, so that the fungus came to be practically lying in a mass of pus (Pl. III, fig. 1); but in one specimen from the subcutaneous tissue of Mr. Anderson's case the fungus was surrounded by firm granulation tissue (Pl. III, fig. 2), which formed a kind of capsule. This specimen, of which I have serial sections, shows points in the structure of the Actinomyces colony which have not, I believe, been before illustrated. Boström regards the typical colony as a hollow sphere, the shell or mantle of

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which is composed of a dense felt-work of branching threads from which clubs may project outwards, and the cavity of which is occupied by irregularly arranged, sparsely branching threads. At one point the shell is broken, and here the filaments grow into the surrounding tissues. Pl. III, fig. 2, shows clearly this arrangement of the hollow sphere and the opening into the shell, but it also shows, what Boström did not mention, the cavity of the sphere almost entirely filled with small round cells of the granulation tissue. The mycelium is entirely absent from the centre. The peripheral zone is also composed partly of leucocytes, and I was not able in any of the sections to distinguish clubs. It is possible, however, that the indistinct red-staining matter of this zone may be partly composed of club substance.

In the sections of the liver of my father's case I nowhere found this structure of a hollow sphere. The appearances were always such as that shown in Pl. III, fig. 1, a central blue mycelium with radiating threads extending into the pus-cells around, and a more or less homogeneous matrix, in which rarely faint indications of radial striation could be found, which might be composed of club substance, or perhaps in part of the detritus of dead pus-cells. Some of the blue filaments extend beyond this matrix, just as in the fresh spherule they project beyond the clubs.

Serial sections through spherules from the urine of my own case helped, however, to explain these apparently different types (Pl. III, fig. 3).

Each fully developed spherule is a mulberry-like colony of ray groups, which groups may tend to form a kind of mantle at the surface, while centrally they are connected by a more scanty mycelium. Broken-down pus-cells and detritus form a uniting medium for the whole. In the smaller spherules, on the other hand, the central portion is still occupied by ray groups, such a spherule thus having a close resemblance to a segmenting ovum. Growth then takes place at the periphery, and a central cavity is

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left, which is occupied by degenerated products of the tissues of the host.

In regard to the systematic position of Actinomyces, the facts mentioned tend, so far as they go, to support the doctrine of Boström, that it is not one of the higher fungi, but one of the schizomycetes, probably one of the pleomorphic bacteria allied to Cladothrix.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 16.)

DESCRIPTION OF PLATES II AND III.

Actinomycosis Hominis of the Urinary and Alimentary Tracts. Recovery. (Dr. W. B. RANSOM.)

PLATE II.

FIG. 1.—Part of an Actinomyces spherule examined in the fresh urine of W. K—. ($\frac{1}{6}$ inch.)

FIG. 2.—Clubs treated with 5 per cent. NaCl solution, showing fractures and the axial thread. ($\frac{1}{12}$ inch.)

FIG. 3.—Threads breaking up into rods and enclosing spore-like bodies, from Dr. W. B. Ransom's case, W. K—. (Methyl violet, $\frac{1}{12}$ inch.)

PLATE III.

FIG. 1.—Actinomyces at margin of hepatic abscess in Dr. W. H. Ransom's case. (Gram, alum-carmine, and eosin; $\frac{1}{12}$ inch, reduced.)

FIG. 2.—Section of granulation tissue of cheek of Mr. Anderson's case, showing the arrangement of the filaments as a hollow sphere broken at one point and filled with granulation tissue. Clubs are not visible. (Gram and Rubin; $\frac{1}{6}$ inch.)

FIG. 3.—Section of an Actinomyces spherule from the urine of W. K—. (Gram and Rubin; $\frac{1}{12}$ inch.)

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Fig. 2.

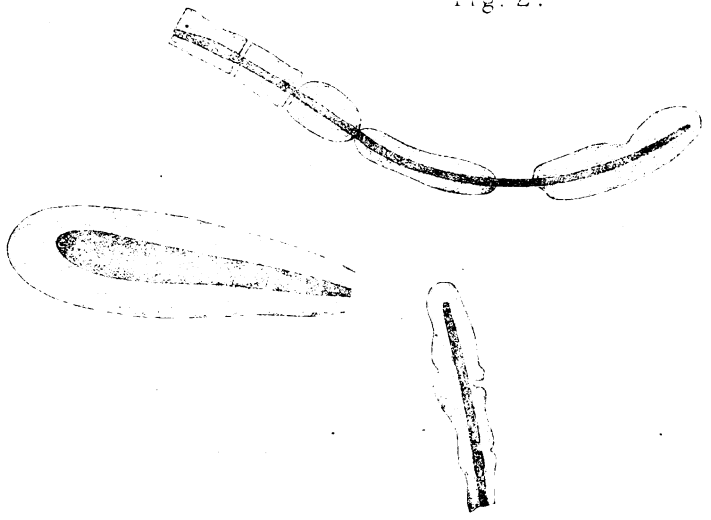

Fig. 3.

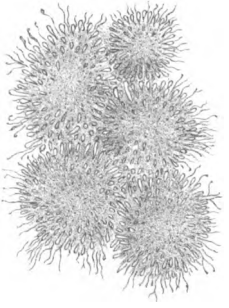

Fig. 1.

W. B. R. del. Mintern Bros. lith.

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Fig. 3.

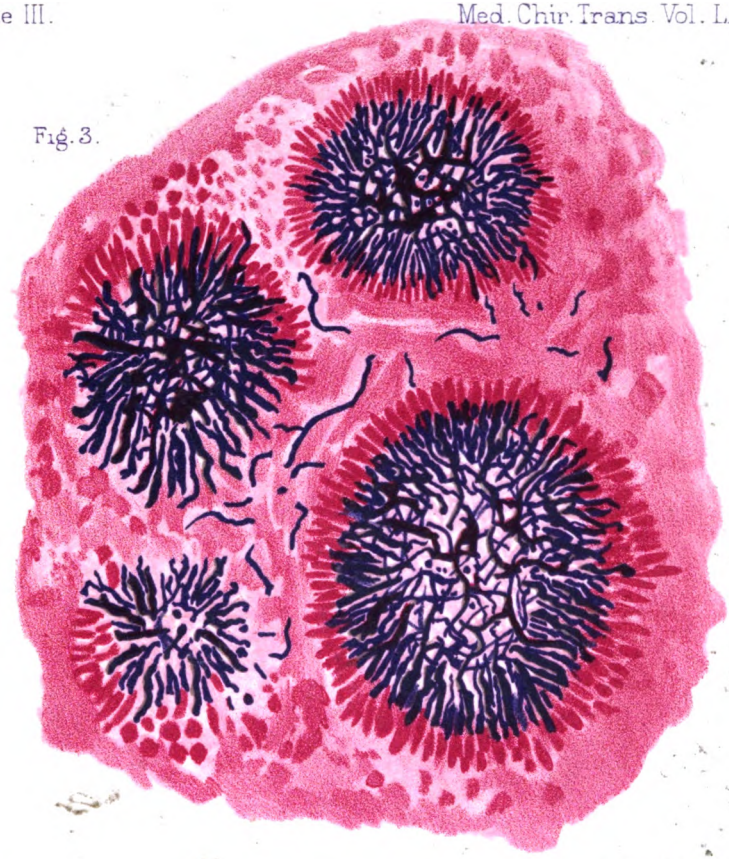


Fig. 2.

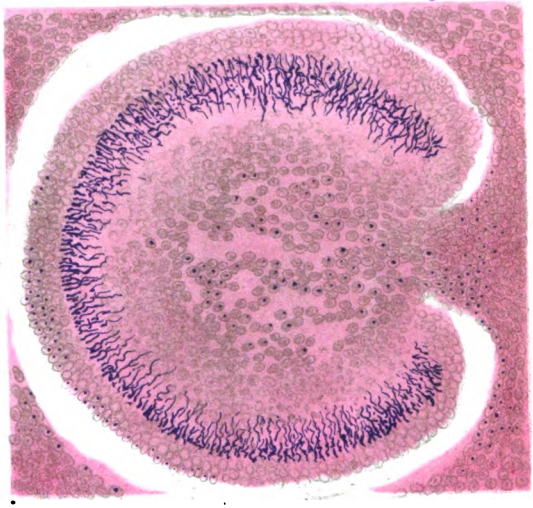
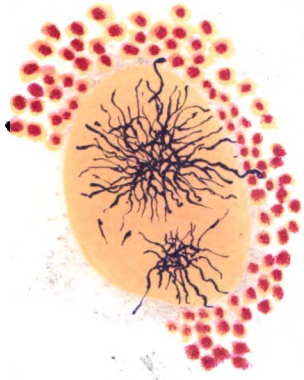


Fig. 1.



W.B.R. del.

Mintern Bros. Chromo.

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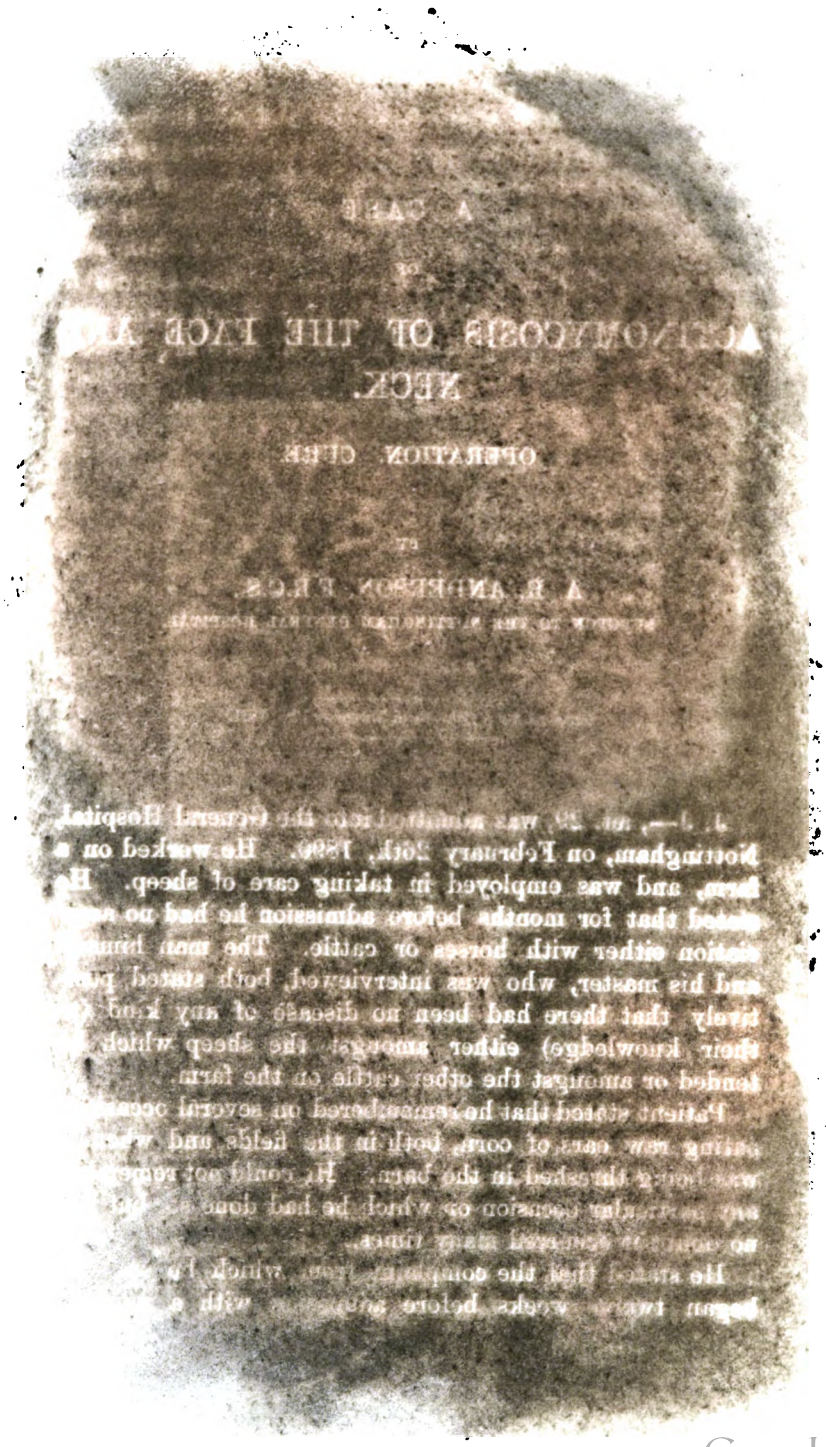

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A CASE

OF

ACTINOMYCOSIS OF THE FACE AND NECK.

OPERATION. CURE.

BY

A. R. ANDERSON, F.R.C.S.,

SURGEON TO THE NOTTINGHAM GENERAL HOSPITAL.

Received April 13th—Read November 10th, 1891.

J. J—, æt. 29, was admitted into the General Hospital, Nottingham, on February 26th, 1890. He worked on a farm, and was employed in taking care of sheep. He stated that for months before admission he had no association either with horses or cattle. The man himself and his master, who was interviewed, both stated positively that there had been no disease of any kind (to their knowledge) either amongst the sheep which he tended or amongst the other cattle on the farm.

Patient stated that he remembered on several occasions eating raw ears of corn, both in the fields and when it was being threshed in the barn. He could not remember any particular occasion on which he had done so, but has no doubt it occurred many times.

He stated that the complaint from which he suffered began twelve weeks before admission with a painless

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swelling of the left side of the face, first noticed over the horizontal ramus of the lower jaw near the angle. From thence it gradually spread up towards the eye and down into the neck. It did not incommode him much, and he was able to work, sleep, and eat (for a time) as usual. After the swelling had been in progress for six weeks he consulted Dr. Wright of Bottesford, who found a collection of pus over the angle of the jaw, which he opened.

The swelling was poulticed, but increased rather than diminished in size, and shortly afterwards another collection was opened, and as no improvement took place he was sent to the hospital.

On admission the whole of the left side of the face and neck was much swollen, and the eye almost closed. Scattered over the face and side of the neck, between the

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margin of the orbit above and the clavicle below, were eight openings, all discharging pus, through each of which a mass of firm material resembling granulation tissue exuberantly fungated.

On passing a probe, bare, roughened bone could be felt through all the openings situated over the face. The skin for a considerable distance round the seat of disease was bright red in colour, much thickened, and had a firm brawny feel—especially on the face, where this condition extended well up into the temporal region. His teeth were very irregular, and on the left side there were carious teeth in both upper and lower jaws.

He did not feel ill, but complained chiefly of not being able to open his mouth or to masticate solid food, for which he had an appetite. He took fluid food well. There was no rise of temperature.

The various openings discharged pus freely, which contained numbers of the pale yellow granules characteristic of the disease, and which were proved by microscopic examination to be actinomycetes.

An examination of the heart and lungs revealed nothing abnormal, and there were no symptoms indicative of disease of any of the other viscera.

On March 4th, after consultation with my colleagues, I decided to remove the disease, or as much of it as I could, and performed the following operation.

Chloroform having been given, an incision was made through the diseased tissues along the lower border of the horizontal ramus of the lower jaw to the angle, then upwards to the zygoma, and inwards along the lower margin of the orbit. The quadrilateral flap of skin thus formed was rapidly dissected up towards the middle line, when the seat and extent of the facial portion of the disease could be well seen.

The facial artery was divided in the first incision and secured, and the oozing of blood from the rest of the cheek, which was at first free, was checked by the ligature of a few small vessels and the pressure of sponges.

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A quantity of the fungating growth lying round and protruding through the openings was scraped away thoroughly, and portions of both the upper and lower jaws and the malar bone, which were found rough and bare, but not otherwise affected, were similarly treated.

A mass of the disease was found extending beneath the malar bone into the zygomatic fossa; this was also scraped out until, as far as could be judged, all had been removed. The masseter muscle, which was obviously affected, and which had been perforated by the disease, was in great part cut away. An incision was then made in the neck along the line of the disease, extending from the ramus of the jaw nearly to the clavicle. The fungating growths were treated in the same manner, and scraped out as rapidly as possible. The operation here was of a simpler nature than in the face, as the disease had not passed beneath the deep fascia.

The facial nerve was not recognised, but was no doubt injured during the operation. The skin was brought together here and there with wire and horsehair sutures, and a dressing of mercurial wool applied.

On March 7th, three days after operation, the following note was made:—"He is doing well. The wounds are healing; there is a free purulent discharge in which the small bodies, which were before present, are not noticeable. The dressings to-day were shaken and washed in a basin of water and the deposit examined carefully, both with the naked eye and with the microscope, but no actinomycetes were found. Thus, although it cannot be said with certainty that they are absent, yet it is evident that they have at any rate become scarce. His temperature has remained normal since the operation."

From this date he made an uninterrupted recovery; the facial wound healed in parts quickly and in others by granulation, that in the neck almost wholly by granulation. No actinomycetes were again found in the discharge, although they were repeatedly and carefully looked for. The brawny swelling and angry redness of the skin

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gradually subsided, but did not completely disappear for months afterwards.

He was discharged on May 21st, two and a half months after operation, being then well, except for two superficial granulating patches which had not healed.

Within a month of his discharge he returned with these wounds healed, and expressed himself as being quite well.

He now (April, 1891) says that his health is perfectly good. He is engaged as an agricultural labourer, and is as well able to do a day's work as ever he was.

One of the chief features of interest in the foregoing case is the fact that the operation proved so readily successful in spite of the wide area involved.

The interesting question at the time from a clinical point of view was, whether the area of chronically inflamed and indurated skin which surrounded and spread away from the fungating sinuses was itself affected with the disease. If so, it was clearly impossible to perform an operation which would completely remove the whole of the affected parts. I believed, however, that even if they were so affected, the fungus if present would probably be sparsely scattered through the surrounding skin, and that if the bulk of the malady were removed the remainder might cease to multiply, and die a natural death.

I removed at the time of operation several portions of the red and thickened skin near the parts undoubtedly diseased for the purpose of determining this point; and Dr. Ransom, who has examined them microscopically, reports that he has failed to find any actinomycetes in them. It was interesting to note how the integument gradually cleared up and resumed a healthy aspect.

The result in this case is such, I think, as to encourage the undertaking of extensive operations for the relief of this disease in accessible parts of the body, even though the operator may foresee his inability to remove the whole of the apparently affected parts.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 16.)

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ON THE

OCCURRENCE OF PLEURAL EFFUSION

IN

ASSOCIATION WITH DISEASE OF THE ABDOMEN.

BY

LAWSON TAIT, F.R.C.S.,

PROFESSOR OF GYNÆCOLOGY IN QUEEN'S COLLEGE, BIRMINGHAM.

Received June 26th—Read November 24th, 1891.

I HAVE devoted as much time as my engagements have permitted during the last few months, and more casually for some time before that, to an examination of the literature of diseases of the chest, to discover if any author has drawn attention to the occasional occurrence of effusion of serum into the pleura as a symptom occurring in diseases of the abdomen. Up to the present, however, I have not met even with a record of an instance of the kind. My own experience is sufficiently large now to make it perfectly certain that in a considerable number of cases pleural effusion occurs, and when it does occur it may be a symptom of very great, indeed of grave importance. My principal object is to draw attention to

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this occurrence, so that others whose special experience in diseases of the chest must be much larger than mine, may lend their assistance to a more complete investigation of this phenomenon.

I have now performed very nearly 3000 abdominal sections. I have seen in addition a large number of cases of disease in the abdomen, where for one reason or another operations have not been performed, or where they have been performed by other surgeons, and therefore I am not in a position to give any statement as to the exact conditions. Indeed, I cannot lay claim to have got an accurate statement of the total number of cases in which even in my own experience pleural effusion has been a prominent feature; but I think that probably I have not had my attention drawn to it, nor have I had occasion to discover its presence in more than twenty cases, and save in five of these cases I can do little more than trust to my memory for the details. But in the fourteen or fifteen cases in which only the ultimate results are accurately recorded, these are so uniform that there could be little doubt in which direction the bulk of my experience points. In the group of cases in which I have detailed records the experience uniformly points in the same direction as in the other fourteen or fifteen, and this is to establish the conclusion that pleural effusion in the presence of an abdominal tumour or of serious abdominal disease is an indication of the very gravest possible kind.

In order that I may make my meaning clear, permit me to give in detail the first case in which my attention was drawn to the symptoms:

On the 2nd of March, 1878, I was summoned down into Merionethshire to see a patient aged twenty-six, under the care of Dr. Price Jones, of Llangollen. She had been married eleven months, and had been confined on the 21st of February of a child, the labour being in every respect ordinary except that the child was stillborn and

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the placenta somewhat friable, but it came away easily. All went well except that the abdomen did not go down completely as usual after labour; indeed, the midwife who attended the labour says that it diminished very little in size after the confinement. Symptoms resembling those of peritonitis came on next day, and on February the 28th she is reported as presenting a sunken, dejected appearance, pulse 100 and temp. 99°, and abdomen immensely distended, with a uniform dull note all over. The motion of the diaphragm was impeded, so as greatly to interfere with breathing. I saw her on the morning of March the 3rd at about 2 a.m., and found the above condition increased; pulse was 180, and breathing 50 to the minute; the patient was extremely exhausted, the uterus was fixed high up in front, and associated intimately with a large bulging tumour which occupied the whole abdomen. In order to save her from threatened suffocation I tapped her above the fundus of the uterus, and removed nearly five quarts of what seemed to me almost pure blood. This greatly relieved her, so that at seven o'clock on the morning of the 3rd her pulse had fallen to 120, and respiration to 32. There was left in the abdomen a large doughy mass, which seemed to me evidently to contain blood-clot. She improved, however, during the next few weeks very rapidly, and she was removed to Birmingham and admitted to the Hospital for Women on March the 30th, but the journey had evidently been a great deal too much for her, as next day acute œdema of the legs came on, and on April the 2nd the abdomen was again so distended as to induce great dyspnœa, and made it necessary for me to tap again. On this occasion I removed eight and a half pints of a fluid, half blood and water. The patient was greatly relieved, but in the afternoon of the 3rd of April dyspnœa was again so urgent that Dr. Heslop saw her in my absence, and recognising effusion into the chest, recommended that I should tap the left pleura at once on my return. This I did, and I removed three and a half pints of bloody serum; on the 4th I removed two and a half

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pints of bloody serum from the right pleura, and we came to the conclusion that there was malignant disease in the chest. The effusion, which must have occurred very rapidly, did not recur, and the patient's sufferings from abdominal distension were so great that on the 6th I determined to perform abdominal section, and in this I had the advantage of the assistance of my colleague, Dr. Savage, and the aid and advice of Dr. Marion Sims, who was my guest at the time.

I made a median incision five inches long, and passed through the ordinary textures and then through a jelly-like layer, which was undoubtedly a mass of malignant disease, and then came into a large cavity lined with thick layers of firm blood-clot. We did not think it desirable to disturb this, nor to separate what looked like a cyst wall from the abdomen, on account of the possibility of unrestrainable hæmorrhage. A drainage-tube was inserted and the wound closed. She did fairly well during the 7th and 8th of April, but on the evening of the 9th bilious vomiting came on, and she died about 5.30 p.m.

A post-mortem examination was made by Dr. Saundby on the 10th of April, and he favoured me with the following report:

The body was that of a young woman fairly developed, emaciated, yellow fluid issuing from mouth; a partially healed linear incision ran from the umbilicus to the pubes; hypostatic congestion and rigor mortis were present. The head was not examined.

The left pleural cavity contained about two quarts of blood-stained serum; the lung collapsed, but healthy in texture. There was a fungating ulcerated growth, about the size of a walnut, covered with blood-clot, on the pleural surface of the diaphragm. The right pleural cavity contained about a pint and a half of similar fluid; lung floating, healthy. There was a growth similar to that on the left side on the surface of the diaphragm, pleura, and also on the ribs, under the parietal pleura. The pericardium

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and heart were healthy, the ventricular cavities full of clots. The posterior mediastinal glands were swollen to the size of hens' eggs by cancerous infiltration.

The lower part of the cavity of the abdomen was filled by a large cystic mass adherent anteriorly to the abdominal wall and to the uterus, broad ligaments, and lower part of rectum. It was removed *en masse*, and on examination was found to consist of a large cystic cancerous tumour of the left ovary, situated behind the uterus and pressing down posteriorly behind that organ and the rectum and extending upwards, and anteriorly till it came in contact with the abdominal wall. It did not involve the coats of the bowel.

The large cyst which had been opened by the incision through the abdominal wall was of varying thickness; its walls were irregular, and in many places covered with blood-clot. Anteriorly it was adherent, but easily separable from the uterus and vagina, the structures of which were not involved. The other ovary was also adherent, but free from disease. The microscopical appearances of the growth were those of encephaloid cancer. There was a cancerous mass in the line of incision of the abdominal wall, and the mesenteric glands were cancerous. All the other organs were healthy.

In the case just narrated the onset of the hydrothorax was probably very rapid; indeed, it wants but little consideration of the case to see that the whole dreadful state of the patient was an example of how rapid cancerous pathological processes may be. The lesson was so fervidly imprinted upon my mind that from that moment I regarded pleural effusion in all abdominal cases as very serious, and I established for myself a conclusion that when the pleural effusion had an infusion of blood in it the only opinion that could be maintained was that the whole disease was due to cancerous development.

The other cases in which this symptom has been brought to my notice have had, so far as any fact in connection with them in my memory serves me, uniformly fatal end-

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ings, and I think I am justified in concluding that they were all malignant; and I believed that this conclusion from the experience just narrated was likely to be accurate in the majority of the cases. Acting upon such an accumulated experience, I had determined in my own mind to discourage all operative proceedings in cases where, as well as abdominal disease, there was ascitic effusion with well-marked pleural effusion on both sides, especially if the latter were determined by aspiration to be of a sanguinolent character, on the ground that the certainty of these cases being malignant was almost absolute, and as the probability was that the pleural lining was infected as well as the peritoneal surface, no hope could be entertained of relieving them by any operation. I have now, however, to place on record a case which shows that such a conclusion may be quite erroneous. The following case displays a remarkable example of a combination of peritoneal and double pleural effusion, the fluid in all three cavities being markedly bloody. I think it is perfectly certain from the result that the disease was not cancerous, and an operation has resulted in a cure absolute and, I trust, permanent.

L. T—, single, æt. 36, was sent to me by Dr. Brown, of Tintern, in January, 1890, with a large abdominal swelling.

Her menses began at the age of sixteen, were never quite regular, occurring only every five to eight weeks (never four weeks), lasting four days, scanty, not painful. She menstruated four weeks before admission. She was always subject to indigestion and irregularity of the bowels, sometimes having diarrhœa, and sometimes obstinate constipation. For five years before admission she had symptoms of irritation of the bladder. She had occasionally retention of urine for many hours, and then suddenly the obstruction would be removed, and she would pass a large quantity of urine. She had no leucorrhœa. About June, 1889, her abdomen was noticed to be enlarged. This

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steadily increased, very rapidly during December, 1889. There was never any pain in the abdomen or back.

For a month before admission she had severe cough, shortness of breath, and night sweats, with profuse expectoration of much black-coloured stuff.

Condition on admission (January, 1890).—Face thin and anxious, body generally emaciated, abdomen greatly enlarged. She breathed rapidly and with difficulty. On examining the chest the left side was absolutely dull nearly up to the clavicle. There was no vocal fremitus or vocal resonance, the intercostal spaces were increased and bulged, the left side of the chest moved but slightly during respiration, and the heart was displaced to the right. The heart was rapid and weak, with its first sound faint. In addition to the evident hydrothorax of the left side there was some bronchitis on the right side.

On examining the abdomen it was found to be greatly distended, the superficial veins were enlarged, and the umbilicus was everted. There were all the physical signs of ascitic effusion free in the peritoneal cavity. In addition there could be felt on deep pressure through the fluid a large rounded solid tumour, apparently moored in the pelvis and floating freely in the ascitic fluid. As the breathing was much distressed the left pleural cavity was aspirated, and ninety-five ounces of blood-stained serum removed. A few days after the tapping of the left side fluid was discovered in the right pleura, and was similarly removed. It contained a quantity of blood. The diagnosis of malignant disease of the peritoneum with secondary infection of the pleural surfaces was made. I declined to operate on the abdominal tumour, and the patient returned home to die.

About a fortnight after returning home her left pleura was again tapped, and eighty ounces of pale yellowish fluid removed. After this she had a slight attack of inflammatory pleurisy. In February, 1890, the abdomen was tapped, and eleven quarts of pale yellow thin ascitic fluid removed. From this time until February, 1891, her

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abdomen was tapped over thirty times, from eight to fourteen quarts of thin clear yellowish fluid being removed at each operation. She was last tapped on February 28th, 1891, when eleven and a half quarts was removed. The pleural effusions did not recur. During the summer of 1890 her legs became œdematous; but this complication subsided, and when readmitted had quite gone. After each tapping the solid abdominal tumour could easily be felt. Before readmission her general health had greatly improved, her bowels were regular, and her appetite fair. Her urine was scanty, and deposited abundant urates. There was no albuminuria. The patient was readmitted into my private hospital on March 4th, 1891; she was then in much better health. She had no pulmonary symptoms, and she was not so emaciated. The pleural effusion had not reappeared since the last thoracentesis in January, 1890.

On examination there was an impaired percussion note over the base of the left lung behind, and distant breath-sounds. The abdomen was distended with free ascitic fluid, floating in which could be felt the solid tumour before described. It had increased during the interval, but not greatly. It felt multinodular and was slightly tender. It was quite solid, and freely mobile from side to side. It appeared to rise out of the pelvis, and reached up to the umbilicus. *Per vaginam* the uterus was small, and did not move with the tumour, which could be felt behind and above it.

There was no œdema of the legs and no albumen in the urine.

Operation on March 5th, 1891.—I opened the abdomen by a short vertical incision in the middle line, midway between the pubes and the umbilicus. As soon as the peritoneal cavity was reached a large quantity of ascitic fluid escaped. The tumour was now discovered to be a large solid growth of the right ovary. It was quite free from adhesions save to the ovary of the other side. The abdominal incision was increased up to the umbilicus, the

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tumour delivered through the opening, and the pedicle—which was formed by the elongated broad ligament—transfixed and tied with the Staffordshire knot. The ovary of the other side was so adherent that it was removed with the tumour, its pedicle being tied also with the Staffordshire knot. But the Fallopian tube on the left side was not removed. The tumour was now cut away, the peritoneal toilet carefully made, a glass drainage-tube inserted, and the wound closed with ten silk sutures. The patient rallied well from the operation. Very little fluid came through the drainage-tube, and at the end of twenty-four hours it was removed. The bowels moved spontaneously on the third day. Six sutures were removed on the sixth day, and the remaining four on the eighth day. She developed urticaria on the eighth day, affecting chiefly the arms and the abdomen. About the fifteenth day her evening temperature began to rise, and she had pain in the back and the pelvis. On examination a tense rounded fluctuating swelling was discovered behind the uterus. On the twentieth day this was aspirated *per vaginam*, and twelve ounces of thick offensive grumous pus removed. The febrile symptoms subsided but recurred five days later, and the suppurating cavity refilled. It was aspirated on the twenty-sixth day when five ounces of pus were removed. Three days later the pus again accumulated. This time, however, it burst into the vagina spontaneously, and continued to discharge for about a week. After this her recovery was uninterrupted but slow. The exudation mass behind and to the right of the uterus slowly became absorbed, and when the patient left the hospital six weeks after the operation it had almost entirely disappeared. The abdominal incision healed by first intention.

The tumour weighed 2 lbs. 2 oz. It was nearly globular in form and quite solid. Attached to it were the ovary of the other side and the Fallopian tube of its own side. Microscopic examination showed it to be a fibroma. On its free surface were a few small cysts containing clear fluid.

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The lesson in this case is, in my opinion, a very valuable indication that no set of conditions in the abdomen, however apparently unfavourable, are sufficient to justify us in an absolutely unfavourable condemnation in any particular case. Looking back upon my experience of pleural effusion as complicated by abdominal disease, which I have said probably gives the somewhat insignificant number of twenty cases out of more than three thousand, I think that probably my general impression that it is a very fatal complication, especially when the fluid is of a bloody character, is correct; and if half of the cases had been submitted to abdominal section, simply for the purpose of exploration and removing the bulk of fluid, the likelihood is not great that permanent benefit would have accrued in many of them; but if one of the lives had been saved by the discovery of a mistake, I think it would have quite justified the performance of the incision in all the rest, for under such circumstances the mere opening of the abdomen has risk very little, if any, greater than the process of tapping, which has to be employed for the purpose of giving the patient relief. Tapping, however, has the disadvantage that it leaves the condition of diagnosis quite as imperfect as it was before the operation, and I have never in a single instance seen anything like a curative effect from the process of tapping in the abdomen. Even in the successful case I have given in detail, tapping of the pleura did not seem to have the controlling influence preventing further secretion; whilst in the abdomen it had no curative influence at all, having had to be repeated over thirty times. The striking results obtained in this case by the correction of my initial mistake have gone a long way to confirm me in the advisability of extending the principles of exploratory and confirmatory incisions in abdominal disease to an almost universal application.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 23.)

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AN ANALYSIS OF SEVENTY-TWO CASES

OF

UNUNITED FRACTURE OCCURRING IN THE LONG BONES OF CHILDREN.

BY

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Received June 30th—Read December 8th, 1891.

The subject of ununited fractures in long bones is one which has always been of great interest to the surgeon, though he has never been able to give a satisfactory explanation of the cause. During the present century three well-known series of tables of ununited fractures have been compiled. The earliest is by Dr. George W. Norris, a surgeon to the Pennsylvania Hospital, who in 1842 published 150 cases of non-union after fractures.[1] The second series was collected by Dr. Gurlt, and is published in his 'Handbuch der Lehre von den Knochenbrüchen,' Berlin, 1862. It consists of 484 cases drawn from all sources. The third and most extensive series was issued by the late Prof. Agnew, who, in his great work

[1] 'The American Journal of the Medical Sciences,' N. S., vol. iii, p. 1.

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on the 'Principles and Practice of Surgery,' Philadelphia, 1878, has collected, by the energy of Dr. Frank Muhlenberg, no less than 685 cases of ununited fractures of the shafts of long bones. In this large number, however, are included many of Norris's and Gurlt's cases. We have thus in an accessible form 1319 cases of ununited fracture duly tabulated, and including persons of all ages.

No one hitherto has been at the trouble to collect and to publish cases of ununited fracture in children, although it is known that they often possess features of especial interest. As my attention has recently been called to the fact of their occurrence, and circumstances have led me to collate the literature of the subject, I have thought that it might prove useful if I tabulated the cases which I have met with. I have accordingly done so, and it is with a consideration of this table that I propose to occupy a short period of your time this evening.

Sir James Paget, in the very interesting series of essays entitled 'Studies of Old Case-books,' which has been recently issued, says, "I have seen only three cases of ununited fractures in young children, and the measures which are usually sufficient for the cure of this defect in adults were in all these cases completely useless. Similar cases have occurred to others; and, so far as I know, they have not been explained" (p. 130). The table of cases which I present to you shows that Sir James Paget's opinion holds true even when his conclusion is applied to a very large series of cases. How little attention has been bestowed upon the subject of ununited fractures in children is manifest from the fact that it has escaped the notice of so careful and experienced an observer as our President, Mr. Timothy Holmes.

There is no doubt whatever about the rarity of cases of ununited fracture in young children, though I am rather inclined to think that they are becoming somewhat more numerous than was formerly the case. Dr. Norris, in his table of 150 cases, only records a single instance of non-union in a child which had come under his personal

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notice (No. 23). Gurlt in 484 cases gives 14 in children, whilst Agnew in his 685 cases records 28 instances of non-union in children under ten years of age. I find, however, that Prof. Agnew's tables make three separate cases out of one which was recorded by Tamplin, so that the cases which he tabulates as Nos. 641, 642, and 643 are the same patient at different periods; and the same error has crept in with regard to Nos. 65 and 220, so that this reduces the number to a total of 25.

From the annexed table it will be seen that I have been able to obtain details of 72 cases of ununited fracture in the long bones of children under ten years of age. From a consideration of the tables it is obvious that the fractures group themselves into three classes. The first, in which the fracture is intra-uterine, or at any rate in which it was noticed directly after birth; the second class embraces those fractures which occur in young children, often as a result of slight violence, and it is to this class that I wish to direct special attention this evening. Finally, there is a third class which embraces the bulk of the cases in which the fracture took place in older children, either as the result of an accident in the usual manner, or the non-union followed upon an osteotomy or other operation performed for the relief of deformity. As an extremely rare condition spontaneous fracture is met with in children, just as it is occasionally seen in adults. In my table I have endeavoured to exclude cases of non-union following compound fracture, as in such cases the pathology of repair is so different.

Of the cases I have collected it will be seen that 6 occurred in the clavicle, 7 in the humerus, 12 in the femur, and 45 in the leg, where the fracture involved one or both bones. It is interesting to notice that I have met with no recorded case of ununited fracture of the lower jaw, nor as yet of the bones of the forearm,[1] and this is

[1] Since this paper was written Mr. Anderson has had a case of ununited fracture of the radius in a boy under his care at St. Thomas's Hospital. He tells me that after operation he has obtained bony union, and he has kindly

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still more interesting when we consider the statistics of fractures as they are met with in children. I have amalgamated the figures given by Marjolin, Langenbeck, Packard, and Beck, which amount in the aggregate to 1070 cases. Of these there were—

Fractures of the	forearm	. . .	328
,,	,, humerus	. . .	228
,,	,, clavicle	. . .	227
,,	,, femur	. . .	213
,,	,, leg	. . . .	74

—thus showing that although fractures of the forearm are so much more frequent than those of any other bone, they most rarely result in non-union (a very unexpected result at which to arrive), whilst after fractures of the leg the proportion of cases of non-union is appallingly great.

As regards the sex of the patients pseudarthrosis occurred in 40 males and in 29 females, whilst in 3 cases the sex is not mentioned. It seems to be a pretty generally entertained opinion that non-union is more frequently seen on the left than upon the right side. I am sorry that my tables do not settle this point, owing to the fact that so few observers have noted the side upon which the fracture occurred. In 47 cases where the side is mentioned I find that in 24 cases it was upon the right, and in 22 cases it was upon the left, whilst in 1 case both bones of both legs were fractured. Of the total number of 72 cases of false joint bony union resulted in 21 cases owing to the treatment which was adopted; in 4 cases the patient was improved, or in other words the fibrous union was rendered somewhat firmer, thereby enabling the patient to get about in some form of apparatus; but in 45 cases, or nearly two thirds of the total number, the condition of the patient's limb remained the same, and I there-

allowed me to include the case in my table No. 14. I have also found a case of ununited fracture of the radius and ulna recorded by Dr. Norris, but it seems to have been an instance of delayed union rather than one of pseudarthrosis.

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fore class these cases as failures. Of the cases which were cured, nine appear to have been examples of delayed union rather than of ununited fractures, as union took place at intervals varying from three to eighteen months after the injury.

It would appear from a study of the table that ununited fractures in children are becoming somewhat more frequent in this country than they used to be. This can no doubt in part be accounted for by the greater care with which such fractures are recorded; partly by the fact that increased facilities for locomotion now allow of such cases being gathered into one or other of the numerous Hospitals for Children which are established in most large towns. Forty years ago such patients would have remained in country districts, where they would only occasionally have come under the notice of practitioners too busy to give them more than a passing thought, and who when they did think of them would only regard them as hopeless cases sooner or later destined for amputation. There is, however, another reason, I think, for the frequency of ununited fractures in children. In 1814 Roux, the great French surgeon, paid a visit to London, where he appears to have devoted his time to making a careful inspection of the hospitals. On his return to Paris he published his observations in an octavo volume of 368 pages, entitled 'Relation d'un voyage fait à Londres en 1814; ou parallèle de la Chirurgie angloise avec la Chirurgie françoise.' In this work, amongst other points, he remarks (page 192), "Il est probable qu'ils voient [*i. e.* English surgeons] très-souvent des consolidations tardives. Je soupçonne enfin que la pseudarthrose, c'est-à-dire la conversion d'une fracture en une fausse articulation, est un accident qu'ils ont plus souvent que nous l'occasion d'observer. C'est pour nous, chirurgiens françois, une chose si rare, de voir une fausse articulation succéder à une fracture, qu'il y a plusieurs années qu'on n'a pratiqué en France l'opération de White, c'est-à-dire, la résection des fragmens de l'os non consolidé, et que depuis

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quelques années aussi qu'un chirurgien de Philadelphie, Physick, a conçu l'ingénieuse idée du traitement de la pseudarthrose par l'interposition et le séjour momentané d'un séton entre les bouts de l'os non consolidé, pour y exciter l'inflammation adhésive, cette opération n'a été pratiquée, que je sache du moins, par aucun chirurgien françois." This increased frequency of the non-union in England Roux attributed to the fact that the roller bandage used here is much more difficult to keep firmly applied than the many-tailed bandages which are commonly used in France. Whatever may be the real cause of this remarkable rarity of non-union in France, there is no doubt that it was maintained for many years. In a lecture given in 1860 at the Hôpital des Enfants[1] in Paris, M. Guersant says, "I have only seen a single case of ununited fracture occurring in a child in the whole of my long experience, and for this child everything was done, but ineffectually, and her leg had to be amputated (No. 69). M. Marjolin, in his edition of 'Coulon's Treatise on Fractures in Children,' states that up to the time he was writing he had never met with a case.

It appears to me that want of rest is the main cause of the non-union of fractures, though there may be many subordinate factors. Hamilton, in his paper published in the 'Buffalo Medical Journal'[2] so long ago as 1854, pointed out that hinge-movements between the ends of a fractured bone are the most fertile source of non-union. It is exactly this kind of movement which occurs in the broken bones of children when they are nursed or carried about, especially when, as frequently happens, the fracture has been overlooked, and no restraining apparatus has been employed for a week or ten days after the injury. This explanation, however, will not account for the increasing frequency of false joints in children; but I am inclined to correlate this phenomenon with the decadence of bandaging in England. With a roller bandage well

[1] 'Gaz. des Hôpitaux,' 1860, p. 346.

[2] See 'A Practical Treatise of Fractures and Dislocations,' ed. 7, p. 287.

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applied, and a wooden splint, it is quite possible to render all fractures of long bones absolutely immovable, without exercising any injurious pressure. Of late years, however, we are, I think, inclined to trust somewhat too much in cases of fracture, and especially in the fractures of children, to plaster-of-Paris splints and cases, which, being left to others to apply, are not quite accurately moulded to the limb. The increased frequency of non-union is, perhaps, also partly to be explained by the comparative contempt with which we are in the habit of regarding fractures since osteotomy has made us more familiar with their pathology and treatment. This, again, may lead us to be a little careless in insisting from the beginning upon the maintenance of that absolute immobility of the fragments which is so necessary for repair. Then, when fibrous union results, we find to our cost that there is little or no tendency to produce bone, even when we use every means in our power to evoke a deposition of callus. I am utterly unable to explain the pathological process upon which this failure to produce bone really depends, but I cannot help thinking that it is due more to a local than to a constitutional cause, for the failure to unite often occurs in the bones of children who otherwise appear to be in perfect health.

Gentlemen, my purpose this evening has been fulfilled if I have directed your renewed attention to the subject of fractures in children, and if I have pointed out once more how important it is to secure by every means in our power immediate bony union, always bearing in mind that if this process fail our little patient will in all probability be a cripple for life.

In compiling the following table I have placed myself under great obligations to the surgeons of St. Bartholomew's Hospital, to Mr. Pick, Mr. Clutton, Mr. William Anderson, and Mr. Stephen Paget, to each and all of whom I hereby offer my best thanks for the kind permission they have so readily accorded me to make use of their cases.

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Table of Cases.

No.	Sex.	Age of patient.	Bone.	Position.	Side.	Duration.	Treatment.	Result.	Surgeon.	Where recorded.
1	F.	15 mos.	Clavicle	Junction of outer with middle third	Right	4 mos.	Resected and wired	Bony union	Pollard	'Brit. Med. Journ.,' vol. i, 1887, p. 676.
2	F.	22 mos.	,,	,,	Left	8 mos.	Rubbing, firm bandage	Fibrous union	Power	Unpublished.
3	F.	2 years	,,	Not stated	Right	3½ mos.	Plaster-of-Paris bandage	,,	Nairn	Victoria Hospital for Children, 1891. Cf. No. 7.
4	M.	8 years	,,	Middle third	Not stated	Not stated	Not stated	,,	Holmes Coote	'Trans. Med.-Chir. Soc.,' vol. li, p. 146.
5	F.	9 years	,,	,,	Right	Unknown	None	,,	Edgar Willett	Unpublished.
6	M.	12 years	,,	Not stated	,,	Almost since birth	Resection, wired	Bony union	Barker	'Trans. Clin. Soc.,' vol. xix, p. 104.
7	F.	2 years	Humerus	Middle	Left	3½ mos.	Plaster-of-Paris bandage	Fibrous union	Nairn	Victoria Hospital for Children, 1891. Cf. No. 3.
8	M.	5 years	,,	,,	Not stated	12 mos.	Ends resected	Bony union	Brinton	Agnew,[1] No. 172.
9	M.	6 years	,,	Not stated	Left	3 mos.	Tinct. Iodi locally; lime internally for a very rickety child	,,	Paul	'Conservative Chirurgie,' p. 291; Agnew,[1] No. 42.
10	F.	6 years	,,	Lower third	Right	18 mos.	Ends resected and pinned	,,	Langenbeck	'Deutsche Klinik,' 1854, vol. vi, p. 264; Agnew,[1] Nos. 65 and 220.
11	M.	9 years	,,	Junction of middle with lower third	Not stated	27 weeks	Resection; ends drilled and wired	Not stated	Russell	'Lond. Association Med. Journ.,' June, 1854; Agnew,[1] No. 182.
12	M.	9 years	,,	Middle	,,	6 mos.	Ends resected	Bony union	White	'Philosophical Trans.,' vol. li, part 2, p. 657; 'Cases of Surgery, with Remarks,' Lond., 1770, part 1, p. 69.

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13	M.	10 years	„	Not stated	Right	Not stated	Not stated	Fibrous union	Sir Wm. Savory	St. Bartholomew's Hospital, 1869.
14	M.	7 years	Radius	Lower third	Left	38 days	Resected and wired	Bony union	W. Anderson	St. Thomas's Hospital, 1892.
15	M.	12 years	Radius and ulna	Near the middle of the forearm	Not stated	4 weeks	Fixed securely	„	Norris	'Amer. Journ. Med. Sci.' (1839), vol. xxv, p. 273.
16	M.	15 mos.	Femur	Junction of lower with middle third	Left	2½ mos.	Fixed securely	Fibrous union	v. Bruns	Gurlt,[2] No. 132.
17	F.	15 mos.	„	„	„	3 mos.	„	„	Pick	Victoria Hospital for Children, 1891.
18	F.	16 mos.	„	Not stated	„	8 mos.	„	Improvement	Clutton	Victoria Hospital for Children, 1888.
19	M.	18 mos.	„	Junction of middle with lower third	„	11 weeks	Interposed mass forcibly broken up	Fibrous union	v. Bruns	'Deutsche Klinik,' 1861, vol. xiii, p. 172, case 19; Agnew,[1] No. 458.
20	F.	19 mos.	„	Junction of upper with middle third	Right	Congenital	Securely fixed	„	Langton	St. Bartholomew's Hospital, 1890.
21	—	2½ years	„	Not stated	Not stated	5 weeks	Gutta-percha thigh-piece and outside splint for 59 days	Bony union	Not stated	'St. Thomas's Hospital Reports for Patients for 1864,' p. 135.
22	M.	3½ years	„	Multiple spontaneous fractures	Left	6 mos.	Securely fixed	Fibrous union	Pick	Victoria Hospital for Children, 1889.
23	M.	4 years	„	Middle	Right	Congenital	Wired	Bony union	Wutzer	Gurlt,[2] No. 455.
24	—	11 years	„	„	„	4 years	Not stated	Fibrous union	Taylor	St. Bartholomew's Hospital Museum, No. 854.
25	M.	12 years	„	Junction of upper with middle third	„	7 mos.	Ends refreshed	„	Morrant Baker	St. Bartholomew's Hospital, 1890.
26	M.	Not stated	„	Middle	„	11 mos.	Seton: good diet	Bony union	Lyford	'Lond. Med. and Surg. Journ.,' vol. iii (1829), p. 78.
27	M.	„	„	Not stated	Not stated	Not stated	„	„	Brodie	'Lectures on Pathol. and Surgery,' p. 132.

[1] 'The Principles and Practice of Surgery,' by D. Hayes Agnew, M.D., LL.D., Philadelphia, 1878, pp. 752—793.

[2] 'Handbuch der Lehre von den Knochenbrüchen,' by Prof. Gurlt, Berlin, 1862, Th. i, pp. 686—723.

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No.	Sex.	Age of patient.	Bone.	Position.	Side.	Duration.	Treatment.	Result.	Surgeon.	Where recorded.
28	F.	18 mos.	Tibia and fibula	Lower third	Left	Congenital	Securely fixed	Fibrous union	Stephen Paget	Unpublished.
29	M.	4 years	,,	,,	Not stated	,,	Not stated	,,	Curling	'Med. Times and Gaz.,' N.S., xi (1855), pp. 189, 544.
30	M.	4 years	,,	Middle	,,	,,	Resected and wired	Bony union	Wutzer	Gurlt,[2] No. 455.
31	F.	7 years	,,	Lower third	,,	,,	Resection	Fibrous union	Henry Smith	'Trans. Pathol. Soc.,' vol. xviii, p. 215.
32	F.	11 years	,,	Two inches above the ankle	,,	,,	Blister, resection, acupuncture	,,	Mott	'Trans. New York Acad. of Med.,' vol. i (1857), p. 82, case 9.
33	F.	11 years	Tibia	Upper third	Right	,,	Resection	,,	Morrant Baker	St. Bartholomew's Hospital, 1886.
34	M.	21 years	Tibia and fibula	Junction of middle with lower third	,,	,,	Wiring, amputation	,,	Langton	St. Bartholomew's Hospital Museum, No. 858.
35	—	Infant	,,	Lower third	,,	,, ?	Not stated	,,	Not stated	St. Thomas's Hospital Museum, No. 267[a].
36	F.	13 mos.	,,	1½ inches above ankle	Not stated	6 weeks	,,	Not stated	Packard	Keating, vol. iii, p. 1095.[3]
37	F.	14 mos.	,,	Middle, after osteotomy for badly united congenital fracture	Left	6 mos.	Bone grafted	Death from scarlet fever	Clutton	Victoria Hospital for Children, 1888.
38	F.	2 years	,,	1½ inches above ankle	Right	13 mos.	Drilled, wired	Fibrous union 8 years later	Callender	St. Bartholomew's Hospital, 1879 and 1887.
39	M.	Just beginning to run	,,	Middle	Not stated	Treated at once	Fixed, scraped, wired	Amputation 10 years later	Sir Jas. Paget	'Studies of Old Case-books,' p. 133.
40	F.	2½ years	,,	Junction of middle with lower third	Left	18 mos.	Wired	Amputation	Lockwood	St. Bartholomew's Hospital Museum, No. 858[a].

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41	F.	3 years	„	Just below the middle	Right	Since birth	Fixation and resection	Fibrous union	Ed. Owen	'Proc. Royal Med. and Chir. Soc.,' ser. 3, vol. iv, p. 39.
42	F.	3½ years	„	Junction of middle with lower third	Left	31 mos.	Securely fixed, seton, pegged	Amputation at 15 years	Sir Jas. Paget	'Studies of Old Case-books,' p. 130.
43	M.	4 years	Tibia	Middle	Not stated	15 mos.	Resection	Fibrous union	Tapp	'Med. Times and Gaz.,' 1856, vol. ii, p. 347, No. 2.
44	F.	4 years	Tibia and fibula	Not stated	Left	2 years	„	Improvement	Dupuytren	Agnew,[1] No. 498; Gurlt,[2] No. 416.
45	M.	4 years	„	Lower third	Right	30 mos.	Resected and wired	Amputation	W. Adams	Hunterian Museum, Roy. Coll. Surg., No. 813[a].
46	M.	4 years	Tibia	Not stated	Not stated	12 mos.	Resection	Bony union	W. G. Williams	'Western Med. and Surg. Journ.,' Jan., 1852; Agnew,[1] No. 597.
47	F.	4 5/12 years	Tibia and fibula	Middle	„	5 mos.	Resected and wired	„	Ashby & Wright	'Diseases of Children,' p. 497.
48	M.	5 years	Tibia	Not stated	Right	12 mos.	Ends scraped, seton, pressure	Fibrous union	Brodie	'Lectures on Pathology and Surgery,' p. 132; 'Lancet,' 1834-5, pp. 262, 532.
49	M.	Not stated	Tibia and fibula	„	Not stated	Not stated	Securely fixed	„	Jameson	Unpublished.
50	F.	5 years	Tibia	Junction of middle with lower third	Left	2 years	Pegged, resected, wired	Bony union	Fifield	'Med. News,' Philadelphia, vol. xlii (1883), p. 414.
51	M.	5 years	Tibia and fibula	Middle	Right	2 years	Not stated	Fibrous union	Brodie	'Lond. Med. and Surg. Journ.,' vol. vi (1835), p. 667, and vol. vii, p. 443.
52	M.	5½ years	„	A little below the middle	„	4 years	Rubbed, wired, pegged, fixed	„	Sir Geo. Humphry	Cambridge University Pathological Museum, No. 1324.
53	M.	6 years	„	Junction of middle with lower third	„	4 years	Bone grafting from rabbit, twice	Amputation	Southam	'Brit. Med. Journ.,' vol. i (1891), p. 1179.
54	M.	6 years	Tibia	Not stated	Not stated	3 years	Acupuncture	Improvement	Bell	Roux,[4] p. 195; Agnew,[1] No. 584; Gurlt,[2] No. 267.

[3] 'Cyclopædia of the Diseases of Children,' by J. M. Keating, M.D., Edinburgh and London, 1890.

[4] 'Relation d'un voyage fait à Londres en 1814,' par Philibert Joseph-Roux, Paris, 1815.

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No.	Sex.	Age of patient.	Bone.	Position.	Side.	Duration.	Treatment.	Result.	Surgeon.	Where recorded.
55	M.	6 years	Tibia and fibula	Not stated	Left	58 mos.	Twice resected	Bony union	Davies Colley	Guy's Hospital Museum, 1260_{25}.
56	M.	$6\frac{7}{12}$ years	,,	Lowest fourth	,,	$5\frac{1}{2}$ years	Splints and plaster of Paris	Fibrous union	Edmund Owen	'Proc. Royal Med. and Chir. Soc.,' ser. 3, vol. iv, p. 38.
57	M.	8 years	,,	Middle	Not stated	4 or 5 years	Pegged	Amputation 2 years later	Holmes Coote	The 'Lancet,' vol. i (1862), p. 664.
58	F.	8 years	,,	,,	Right	5 years	Resected and wired	Amputation	Sir Jas. Paget	'Studies of Old Case-books,' p. 135.
59	M.	8 years	Tibia	3 inches above ankle	Left	12 mos.	Resection	Fibrous union	Churchill	Victoria Hospital for Children, 1886.
60	F.	9 years	,,	Not stated	Right	Several months	Tenotomy, drilling	Bony union	Dieffenbach	Casper, 'Woch. f. Heilkunde,' 1846, p. 730.
61	M.	10 years	Tibia and fibula	,,	Not stated	3 years	Resection	,,	Stocks	Agnew,[1] No. 493; 'Massachusetts Med. and Surg. Reporter,' 1870.
62	M.	10 years	Tibia	,,	,,	3 years	Not stated	Improvement	Ward	'Medical Times,' vol. xxii (1850), p. 593.
63	F.	10 years	,,	Lower third	,,	8 years	,,	Amputated	Cæsar Hawkins	'Trans. Pathol. Soc.,' vol. ii, p. 253.
64	F.	10 years	Tibia and fibula	,,	Left	8 years	Leather splints	Amputation	Not stated	St. George's Hospital Museum, series i, No. 203; cf. p. 132.
65	M.	13 years	,,	,,	Right	$12\frac{3}{4}$ years	Resected and pegged	Fibrous union	Bowman	The 'Lancet,' vol. ii (1852), p. 153; Agnew,[1] No. 562.
66	M.	14 years	,,	,,	Left	6 years	Resected and pinned	,,	Morrant Baker	St. Bartholomew's Hospital, 1889.
67	M.	Not stated (boy)	,,	,,	Not stated	18 mos.	Ends bored and scraped	Tibia, bony union; fibula, ununited	Wood	'American Med. Times' (New York), 1861, vol. ii, p. 13.

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68	M.	Not stated (boy)	Tibia	„	Left	4 years	Seton	Fibrous union	Lawrence	The 'Lancet,' vol. ii (1857), p. 198.
69	F.	Not stated (girl)	Tibia and fibula	Not stated	Not stated	Not stated	Fixation, cautery, seton, rubbing	„	Guersant	'Gazette des Hôpitaux,' 1860, p. 346; Coulon, 'Traité clin. et prat. des fractures chez les enfants,' p. 49.
70	F.	20 years	Tibiæ and fibulæ	„	Right and left	Since childhood	Not stated	„	Marshall	'Trans. Path. Soc.,' vol. xviii, p. 240.
71	M.	23 years	Tibia and fibula	Lower epiphysial line	Left	19 years	Wired 11 months before death	„	Sir Geo. Humphry	Cambridge University Pathological Museum, No. 1325.
72	F.	25 years	Tibia	Lower third	Not stated	23$\frac{10}{12}$ years	Firm fixation	Bony union	Tamplin	'Lond. Med. Gaz.,' vol. xlvi (1850), p. 140; Agnew,[1] Nos. 641, 642, and 643.

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Appendix.

By the kindness of the authorities of St. George's Hospital in general, and of Dr. H. D. Rolleston in particular, I am able to show a dissected specimen (No. 64 in the table of cases) of an ununited fracture in the left leg of a child. The tibia is broken about four inches and the fibula about two inches above the ankle. The lower fragments of both bones are tilted forward, overlapping the opposite fractured extremities. The upper ends of the lower fragments are rounded off, and are covered with a dense fibrous structure. The lower ends of the upper fragments are connected with the contiguous fragments throughout nearly the whole of the circumference by a dense fibrous capsule or band holding them firmly together. A part of each upper fragment, however, is not covered with any such deposit, but is movable upon a similar uncovered part of the lower fragment. The tibialis anticus and the extensor proprius hallucis are pushed outwards by the projecting part of the tibia, and the extensor longus digitorum is displaced by the fibula, which is embedded more or less in its muscular fibres. The peroneus longus and brevis are also thrown forwards and outwards as they pass behind the external malleolus.

The fracture is said to have been of eight years' standing in a girl of ten years old. The bones were movable at the seat of fracture, and the skin was ulcerated at the same point, owing apparently to the projection of the fragments. The leg was wasted, and was three inches shorter than its fellow.

When the child was two years old she was struck with a cricket ball, which bruised but did not appear to break her leg. She was put to bed and poulticed, and after a time leather splints were applied. She was at this time in bad health, and her leg became "bowed out." Ten months later it broke, and since that time it has twice

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been broken. Two years before her admission to the hospital she was an in-patient for six weeks, when splints were applied, but without good result. The limb was amputated, and the patient made an excellent recovery.[1]

[1] St. George's Hospital Museum, Series I, No. 203.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 35.)

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ON TETANUS AS A COMPLICATION OF OVARIOTOMY.

BY

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Received October 7th, 1891—Read January 12th, 1892.

This formidable disease has many times been treated of as a complication of surgical cases and the lying-in state, but only twice before has it received any individual attention when connected with ovariotomy. Theophilus Parvin[1] relates such a case, and gives a table of thirteen others; while Olshausen[2] quotes thirty-eight cases and devotes a few lines to the discussion of this complication. With a knowledge of these facts, and having had the misfortune to meet with a case in my own practice, I felt that sufficient excuse existed for bringing this subject before the Society.

My first object was to obtain, if possible, the statistics of ovariotomies (excluding hysterectomies) from the various specialists in Europe and elsewhere, and their opinions as to the causation and pathology of tetanus. I accordingly addressed a series of questions to the more important university towns in Europe, sending out 81 letters, viz. to Germany 38, Austria 12, Italy 10, Switzer-

[1] 'American Gynæcolog. Trans.,' 1877, vol. ii, p. 317, and Chart, pp. 322, 323.
[2] 'Krankheiten der Ovarien,' 1886, s. 369.

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land 7, Russia and France 6 each, and Belgium 2. I received 68 answers, 28 of which were of little or no importance, giving only negative evidence; I found, however, that 40 were of extreme value, being written by men of high repute and of undoubted experience. Many of them returned long written opinions on the matter, which I have incorporated in the following paper.

Letters were also addressed to 67 operators in Great Britain and Ireland, viz. in London 30, England 16, Scotland 11, and Ireland 10: as a result I received 48 answers, containing much additional evidence. The replies from America and Australia were so meagre and unsatisfactory, that I have confined my investigations to Europe.

The statistics as obtained above, together with the results of a minute search through the literature on the subject, have enabled me to collect 64 cases, of which fairly full details have been given in 57 in Table A, while the 7 other cases are given in the appendix, more complete details not being obtainable.

I will first relate a few particulars of the case which occurred under my own care, and then proceed to a review of the whole number.

Mrs. B—, married, 2-para, æt. 35. I first saw her in consultation with my friend Mr. H. F. Bailey, of Blackheath, on September 8th, 1888, when she had symptoms of an anomalous character with emaciation and irregular and profuse menstruation. The only physical sign then discovered was a thickening in the posterior cul-de-sac with impaired uterine mobility.

December 27th, 1888.—The thickening had increased, and was evidently the base of a swelling of indefinite size; the uterus was fixed and ascites present; progressive emaciation.

January 7th, 1889.—On opening the abdomen about half a gallon of yellowish-green ascitic fluid escaped; a villous mass about the size of a cocoa-nut with a hardened base was found in the posterior cul-de-sac; it broke

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away easily when handled, and was whitish in colour. The intestines and surfaces of the right broad ligament were studded with similar small growths about the size of peas. The ovaries were apparently healthy; as much as possible of the new growths was removed by the hand and sponges, and the abdomen closed. The patient made an excellent recovery, put on flesh, and was able to walk three or four miles daily without fatigue.

October 2nd, 1890.—The patient was seen again. The thickening in the posterior cul-de-sac had quite disappeared, but a hardish rounded mass the size of an orange, mobile, and not attached to the uterus, could be felt occupying the right side of the pelvic cavity.

January 21st, 1891.—The patient's general health has begun to suffer, the swelling has increased rapidly, and ascites is present. On opening the abdomen ascitic fluid, similar to that observed in the first operation, escaped. The tumour was a multilocular cyst, each cyst containing papillomatous growths; there were dense adhesions, especially to the abdominal parietes; three of the cysts were tapped, permitting the escape of a thick creamy fluid, mixed with sago-like bodies; the pedicle was broad and thick, and was tied in four sections with carbolic silk ligature; the stump oozed a little, and a second ligature was passed encircling the whole pedicle. In the right broad ligament, near the uterus, a swelling the size of a pigeon's egg was found; on incising the capsule, sago-grain material escaped. A glass drain-tube was inserted, and carbolic sutures were used to close the incision.

23rd.—Sickness troublesome, but relieved by Cocainæ Hydrochlor., gr. ½; Acid. Hydrocyan. dil., ℥ij; Aq. ad ℥j, given at intervals.

26th.—Was restless last night; this was attributed to her having seen her husband for a few minutes; at 5 p.m. slight stiff neck and sore throat; in the evening she could not open her mouth as wide as usual. A mixture of chloral gr. xv, and bromide of potassium gr. xxx, was

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given at once by the mouth. This was repeated at intervals by the rectum as the spasm of the masseters increased.

27th.—The spasms have become general, and risus sardonicus is well marked; feeding entirely by rectum. Chloroform inhalations when the spasms become very violent. Extr. Physostigmat. fab. gr. $\frac{1}{6}$ injected subcutaneously every two hours, with no apparent result. Death took place after twenty-six hours' illness.

A partial post-mortem only was allowed; the spinal cord was sent to my colleague, Professor Crookshank, who found nothing noteworthy in the naked eye appearances. The parts were not fresh enough to allow of experiments with the tetanus bacillus.

The stump was perfectly healthy, no peritonitis. No tension on the stitches of the incision wound; the omentum was adherent to the anterior parietal wall, but normal in appearance. No fluid in peritoneal cavity.

Remarks.—The tetanus must here have arisen *de novo;* no case had ever been seen by the practitioners in and around Blackheath, and I can say the same for myself. The operation was done in a house on the top of a hill and surrounded by gardens; several abdominal operations had been performed by myself in the same room during the previous year; they had all recovered, having run normal courses. The strictest antiseptic precautions were taken. I think, then, the questions of sepsis, epidemic, and endemic influence can be excluded.

I am obliged, therefore, to fall back upon three other causes as possible, viz.—

1. The number and firmness of the adhesions broken down;
2. The necessity of the second ligature to the pedicle;
3. The low diet after operation;

—all acting in a patient of extremely nervous habit.

Sir George Humphry tells me that he considers the third of these causes a strong factor, and relates his own and Dr. Meadow's cases as instances.

All treatment seemed useless in my case. The rectal

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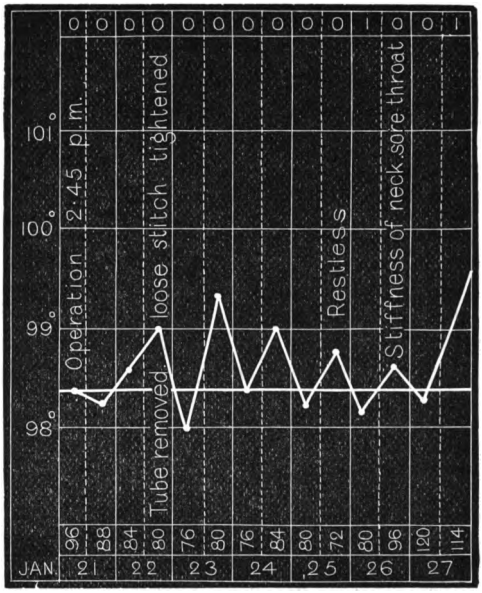
administrations brought on severe spasms, which even the Physostigmatis faba injections did not allay in any way.



TEMPERATURE CHART, showing absence of all indications of sepsis.

I must now call your attention to Table A, where there is a list of 63 other cases of tetanus in addition to my own, with all the more important particulars of each case given; it will be seen that several operators have met with more than one case, viz.—

Stilling	7	†*Kimball	2
Savage	5	Olshausen	2
Thiriar	4	Kaltenbach	2
*Boinet	3	Zweifel	2
*Boye	3	Schroeder	2
Spencer Wells	3	‡*Uhde	2
		Underhill	2

* Operators deceased and no further particulars obtainable.

† Kimball really met with 5 cases in 267 ovariotomies, but particulars of 2 only could be secured.

‡ In 15 cases of ovariotomy.

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I propose to point out any important points in the series of each of these observers and comment thereon.

Stilling's cases (7).—In the 'Deutsche Klinik' Stilling gives a series of 28 ovariotomies, which he describes with the greatest minuteness and wearisome detail, occurring between 1864 and 1872, a period of eight years. Seven of these cases were attacked with tetanus, and six died.

The ninth of this series he performed at the women's Klinik at Cassel, September 2nd, 1865, the clamp treatment being adopted; a normal course ensued, and recovery took place; no signs of sepsis.

The tenth (Table A, Case 6) was on November 6th, 1865, in a private house at Cassel, ran a septic course, and died of tetanus on the twelfth day after operation. This was Stilling's first case of tetanus.

The eleventh (Table A, Case 7) was on August 15th, 1866, nine months afterwards, at the town of Gotha, more than sixty miles from Cassel; ran a septic course, but the patient was convalescent when she was seized with tetanus on the fifteenth day, and died.

The twelfth (Table A, Case 8) was performed seven days later at a private house in Cassel, pursued a normal course, but was seized with tetanus on the ninth day, and died. This was Stilling's third consecutive case.

The sixteenth (Table A, Case 9) did not occur until April 13th, 1868, and in a private house at Oldenburg, at least 150 miles from Cassel. Intense septic peritonitis ensued owing to the stump slipping into the peritoneal cavity; tetanus appeared on the seventh day, and she succumbed in a few hours.

The nineteenth (Table A, Case 11) occurred at Cassel with a similar result.

The twenty-fourth (Table A, Case 13), nearly three years afterwards performed in a private house at Helsa, near Cassel, terminated fatally in a few hours.

The twenty-sixth (Table A, Case 14) was attacked with chronic tetanus, but recovered.

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I have purposely gone somewhat into detail here in order to discover the origin of this appalling series of disasters. How the first arose is not evident; Stilling's previous case was not septic, and there were no other tetanic cases in Cassel at the time. I think the other six are clearly due to contagion; in no other way can one explain the appearance of tetanus at 60 and 150 miles distance respectively from the original site of outbreak. Evidently the hands or the instruments of the operators were at fault, probably the latter.

Dr. Savage's cases (35, 41, 46, 52, 56) are very difficult to understand. In the fifth only was there anything tangible as a cause, and in that at the post-mortem (Case 56) the distal end of the stump was found gangrenous. Long intervals occurred between the cases, and the operations were performed in different localities; moreover during the intervals many other patients recovered without any complications; these five occurred in a series of 1134 ovariotomies.

Thiriar's cases (40, 43, 45, 47).—He himself considers that his first case (40) was caused entirely by the chill; the patient when warm in a heated room was subjected to a draught, and in six hours' time she exhibited signs of tetanus. No history of any chill could be found in the other three cases (43, 45, 47). It will be observed that all the operations were exceptionally easy, there being no adhesions, and the abdominal cavity was open only for a short time. At Nivelles, where the last case occurred, Thiriar found there had been an epidemic of tetanus for two years. It is quite certain that in eighteen months in that neighbourhood there were eight cases of tetanus including Thiriar's, and under four different observers. Brussels, Waterloo, Braine l'Alleud, and Nivelles are all in a line, and not far distant from each other. It appears, however, that by some means Thiriar suspected his hæmostatic forceps as the cause of the mischief; he had these thoroughly disinfected by heat, and never met with a case of tetanus afterwards. Here, I think, is ample proof that

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tetanus was epidemic and possibly endemic in addition to being carried by contagion.

Sir Spencer Wells' cases.—His ninth, twelfth, and thirty-fifth cases (Table A, 2, 3, 4) were attacked with it, and then he had a clear run of 850 cases without an attack.[1] He tells me that about the same time he had a case of tetanus after perinæorrhaphy, and also saw another in consultation arising from a pitchfork wound. The urine of his first patient produced tetanus in frogs when injected under the skin by Richardson.

Underhill's cases (30, 33).—Dr. Underhill operated upon a male patient who had been seized with tetanus, and died on March 8th, 1880. On the 17th he performed ovariotomy on Case 30, who succumbed in like manner; there was no sepsis. Case 33 occurred June 22nd, 1880; the patient was well aware of the existence of the disease, and of the fate of Case 30. The operation was apparently simple, and ran a normal course up to the date of onset of the complication. Another case of tetanus occurred under the same operator in August, 1880. Here, again, contagion was probably the secret of the spread of the disease, although nervous dread in the second case might have been a predisposing cause.

In considering *Zweifel's and Schroeder's cases* (26, 28, 20, and 21) another theory is put forward. Zweifel is of opinion that his cases were infected by the plaster on the newly done-up walls of the operating room. Both occurred during the first half-year after the removal of the Frauenklinik to a newly built pavilion at Erlangen in 1878, and, moreover, they were consecutive cases. Schroeder, his predecessor, experienced the same misfortune. In the autumn of 1874 a new pavilion (not the same as that used by Zweifel) was built for ovariotomies at Schroeder's request; he operated in it, and lost his first two with tetanus.

Zweifel remarks that the Erlangen subsoil consists of a granular dolomite sand, which lies under a considerable

[1] The total number now reaches over 1000 cases.

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layer of garden mould ("humus"). The mortar for the pavilion was formed by digging for the dolomite and mixing it with the lime.

It should be remembered in support of this ingenious but somewhat peculiar theory that the identity of the tetanus bacillus and that found in garden mould (Nicolaier's) was demonstrated in Koch's laboratory, April 10th, 1887.[1]

Olshausen's cases (32, 38) were the ninetieth and hundred and thirty-sixth in a long series, and he is quite at a loss to understand their occurrence except by irregularity of ligature, upon which he lays great stress.

Kaltenbach (17, 18) is himself uncertain about proper ligature in his cases: in one the pedicle was very thick, and not a proper one for the clamp treatment; in the other (17), after falling off of the clamp, a blackish eschar was found, under which was a bright white filament; when this was seized with the forceps the woman, who was an insensitive person as a rule, shrank back in great pain; two days later the first signs of trismus appeared. Nerve irritation was here probably the cause, while contagion was acting in Case 18.

All *clamp cases* necessarily have the bruising, crushing, and tearing elements present, tending to the production of tetanus by nerve-irritation. Olshausen thinks that because 27 out of his 38 collected cases were treated extra-peritoneally, there must be some serious element of danger in the method. There are certain indications that in several of the 27 cases the clamp was either insufficient or unequal. Stilling saw in three of his cases secondary hæmorrhage from the pedicle, which could only be stayed by further tightening. My tables (vide Table D) show that there are more intra-peritoneal than extra-peritoneal cases recorded, viz. 35 to 29, which somewhat weakens Olshausen's theory, although of course a much larger number of operations are performed now than then.

[1] 'Principles of Surgery,' Senn, p. 383.

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In a case related by Rose,[1] at the end of the second week, the pedicle, which had been pierced by a long needle, was twisted tighter and tighter each day; the patient, when apparently convalescent, was seized with tetanus and died. Here the torsion might well be considered as the exciting cause. In many cases it will be found that the omentum was either enclosed in the pedicle, or torn, or over-stretched. Such an injury might without doubt be a source of tetanus from nerve-irritation.

Krassowski's two cases of trismus during operation (Table B) in a series of 128 ovariotomies are of extreme interest, and the cause must have been some part of the operation, *i. e.* tearing of adhesions, or tying of pedicles.

Hennig's case (53), occurring shortly after closure of the wound, might have been due to a similar cause or to the pre-existing gangrene of the tumours.

In Champneys' case (49) tension was the most important cause, the gangrene being secondary.

The "tendency" to tetanus in Thornton's case (37) is of considerable interest, as the actual disease occurred as a sequence of the more formidable operation later on.

Whether *sepsis* is a cause of tetanus or not is, I think, at present uncertain. In the 57 cases given in Table A sepsis was evident during life or post mortem in 7 cases only, it was certainly absent in 33, while no mention is made of its existence or not in 17 cases. Saboïa, of Rio Janeiro, says that before 1877 tetanus was one of the most frequent complications of wounds; since the introduction by him of antiseptics the affection has become much more rare. Dr. More Madden, alluding to trismus nascentium, tells me that in the Dublin Lying-in Hospital from 1757 to 1783, out of 17,650 children born, 2944 died of tetanus, while during the first twenty-five years of the hospital's existence every sixth child died thus. The introduction of antiseptics has entirely changed this. I think we can conclude that sepsis certainly promotes the

[1] Pitha und Billroth, Bd. i, Abtheil. ii, A. Abschn., ii, 5, s. 51.

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spread of tetanus, but that tetanus can arise without any suspicion of sepsis. More than one well-known operator, although entirely discarding antiseptics, has never met with a case of tetanus; while others, who take the most minute precautions in the antiseptic treatment, have had the misfortune to encounter one of these formidable cases.

Low diet after operation seems to be a possible factor in the causation of tetanus. Sir George Humphry tells me that both in his own case (Table A, 1) and that of Dr. Meadows (Addendum to Table A) the diet was of a very unnourishing kind for some days after the operation; this was, I suppose, owing to the former dread of vomiting, and which is now often successfully combated by purgation and nourishing food.

Certain localities appear to have been peculiarly exempt from this disease: according to all the leading operators in Scotland, it has never been met with in that country; the same may be said of Ireland and the south-west of England. Dr. W. J. Sinclair informs me that he has been operating for twelve years at Manchester, and has neither met with a case of tetanus nor heard of one.

Dr. Blanc remarks that, at Bombay, tetanus rages with a greater intensity during the months when the cholera is most fatal; he thinks that tetanus is carried by water in the same way as cholera.

Verneuil[1] has very ably advocated the "equine" origin of tetanus. According to this theory, the earth which causes the disease and which contains the tetanogenic germs is especially that which has received the dejecta of the horse.

The date of origin and duration of tetanus after ovariotomy.—It will be found that 38 cases began on the fifth, sixth, seventh, eighth, or ninth day; while in 2, although the date was certainly one of these, it is not given in the clinical history. Six cases began on the tenth, eleventh, or twelfth day, and 11 cases after the twelfth day. The duration of the disease in the majority of the cases was

[1] "L'origine et la pathogénie du tetanos," 'Rev. de Chirurgie,' 1887, p. 759.

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from one to four days; it was noted as taking a chronic course in 5 cases (2, 7, 10, 14, 17). I think these facts are in accordance with the course of tetanus when complicating any surgical operation.

Although this is essentially a clinical paper, I cannot but allude to the most recent bacteriological researches on tetanus. Kitasato[1] has proved that the bacillus of tetanus can be carried by pus. He found that the bacilli were of a most resistent nature; moist heat at 100° for five months did not destroy them; ten hours' exposure to a 5 per cent. carbolic acid solution left them still very virulent, but they became inert in fifteen hours' time. Three hours in a 1 per cent. solution of sublimate was fatal to them. He also discovered that these organisms are never found at any distance from the wound. It is also well known that the ptomaines of the tetanus bacillus cause tetanic convulsions.[2]

Treatment.—Table A will indicate that every possible remedy was resorted to in the various cases, and with an almost uniform result—failure. It seems to me that the discoveries alluded to above are likely to turn our attention more particularly to the local treatment of tetanus. If the tetanus bacillus causes all the familiar phenomena of tetanus, and if, as is I think proved, this organism is never found far away from the wound, surely our efforts should be first and chiefly directed towards its removal. I believe it is asserted that iodoform possesses the desired germicide power. Certainly at once, on the first symptoms of tetanus showing themselves, all sutures should be removed; and if the pedicle be very thick or broad, it should be examined for a cutting or over-tight ligature. Palliative and general treatment may then be thought of.

The object of this paper has been twofold: firstly, that

[1] "Über den Tetanus Bacillus," 'Zeitschrift für Hygiene,' 1889, Bd. vii, s. 225.

[2] Brieger, in 'Deutsche med. Wochenschrift,' 1887, No. 15, S. 303; and Roswell Park's "Mütter" Lectures, in 'Annals of Surgery,' 1891, vol. xiv, p. 138.

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it might act as a work of reference to future workers; I have, therefore, given facts at full length and in detail: secondly, that it might elicit from others their own experience in a discussion which I trust may result in some definite addition to our knowledge of this terrible disease.

Conclusions.

1. That the operation of ovariotomy has elements in it which are conducive to the production of tetanus by nerve irritation, *i. e.* tearing of adhesions and ligature of pedicle; but that tetanus in these cases does not deviate from the usual series of symptoms which are observed when it complicates other surgical operations.

2. That although some cases of tetanus arise *de novo*, and without necessarily the presence of sepsis, yet the usual manner of spreading is by contagion, and is aided by sepsis.

3. That although the "garden-mould" theory of causation is not established, yet that there is sufficient evidence to recommend avoidance of operating in a room recently plastered, or situated near lately disturbed garden mould.

4. That on the first symptoms of tetanus arising, a strict local search should be made for an irritating cause before proceeding to general treatment.

5. That folding over a broad pedicle which has already been ligatured, and then applying a second ligature, is not to be recommended.

6. That the only means of disinfecting instruments is by boiling them for at least an hour.

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TABLES AND CHART.

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Case No.	Operator.	Hospital or Private House.	Date and Locality of Operation.	Age. M. S. W.	Ch.	Previous History.	Nature of Tumour.	Date of Onset and Termination of Tetanus.
i	G. M. Humphry	P. H.	18, vii, 1855. Barrington, near Cambridge, England	27 m.	3	Tumour in side, rapidly growing 14 months	Left unilocular cystoma, with solid portions	8th day 12th „
ii	Spencer Wells	P. H.	6, x, 1859. London	41 m.	10	Two previous tappings	Multilocular ovarian cyst; 13 pints of fluid, weight 38 lbs.	15th–30th
iii	Spencer Wells	P. H.	28, x, 1859. London	38 m.	?	Markedly hysterical and neurotic	Multilocular ovarian cyst; weight 53 lbs.	7th–10th
iv	Spencer Wells	H.	9, v, 1862. Samaritan Free, London	30 s.	0	Tumour, 6 mos. duration; 2 attacks peritonitis previous	Unilocular ovarian cyst	12th–14th
v	Nélaton	P. H.	17, vi, 1862. Paris	26 s.	0	Tumour, 1 year's growth. Palliative tapping 1 mo. before; refilled in 10 days	Multilocular ovarian cyst; 8 litres fluid	After 14th–21st
vi	B. Stilling	P. H.	6, xi, 1865. Noon. Cassel, Germany	45 m., w. 10 yrs.	4	Tumour, 1½ years, 7 tappings; last 25 days interval; first 8 months before	Left unilocular ovarian cyst; colloid material in pelvis	9th–12th
vii	B. Stilling	H.	15, viii, 1866. Gotha, Germany	33 m.	1	Tumour, 4 years; rapid increase; pain, dyspnœa	Left unilocular ovarian cyst	15th–22nd
viii	B. Stilling	P. H.	22, viii, 1866. Cassel, Germany	35 m.	2	Tumour, 2½ years in left side	Left multilocular ovarian cyst, with colloid degeneration; weight of fluid, 48 lbs.	9th–10th
ix	B. Stilling	P. H.	18, iv, 1868. 9 a.m. Oldenberg, Germany	36 m.	5	Much ascites; tumour, 9 months; tapping 6 days before operation	Large sarcoma, right ovary; left ovary healthy	7th–8th
x	Gilman Kimball	P. H.	2, viii, 1868. Lawrence, Mass., U.S.A.	35 m.	3	Tumour, 1 year; pain excessive; dyspnœa and swollen legs 6–7 weeks	Multilocular ovarian cyst; weight 35 lbs.	10th–20th
xi	B. Stilling	P. H.	27, v, 1869. 5 p.m. Cassel, Germany	56 m.	1	Ovarian swelling 23 years; 37 tappings	Cystoma of both ovaries	7th–9th
xii	C. W. F. Uhde	H.	29, xi, 1869. Braunschweig, Germany	20 s.	0	Irregular menstruation; ascites	Single cyst, right ovary	? –12th

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Operation, Treatment of Pedicle, &c.	Treatment.	Result and Duration of Disease.	Remarks.	Post Mortem.	Reference.
Incision 3 inch, then 4 inch. Pedicle broad, tied in three places, brought out at wound; no clamp	Extr. Belladon., gr. iv; Ext. Cann. Ind., gr. iv, per rectum; morphia, p. r. n.	D.	Disease commenced with trismus; temp. normal, and pulse under 100 up to attack	Stump healthy; mesentery adherent to wound; no peritonitis	'Association M. J.,' 1856, pp. 121, 123, and Priv. Com.
5-inch incision; four vessels tied in omentum. Clamp; broad pedicle; came away 9th day; ligatures 12th. Harelip pins, abdominal incision	Woorara hypodermic, gr. $\frac{1}{16}$; solution applied to blisters on neck, 6 gr. in all (gr. ii ad ʒj)	R.	4th day of operation slight trismus; reappearance 15th day; confined 13 months after	—	'Diseases of the Ovaries,' 1865, p. 35.
4-inch incision; parietal and omental adhesions; short pedicle, clamped with difficulty	Woorara and assafœtida, dose (?)	D. 56 hrs.	Wound healed first intention; bedding changed during a profuse diaphoresis	One pint of blood (fluid) in pelvis	'Diseases of the Ovaries,' 1865, p. 46.
5-inch incision; intestinal and omental adhesions. Clamp, which came away 3rd day; omentum fixed into wound by 2 needles	Turpentine enemata; $CHCl_3$ inhalation; tracheotomy	D.	Temp. not given; no irritated nerve found in cicatrix	Wound united; pedicle and omentum adherent to scar; no peritonitis	'Diseases of the Ovaries,' 1865, p. 105.
Adhesions with omentum and bowel. Clamp came away 4th day. Silver wire sutures for incision	—	D.	Sutures removed 5th day; purgative 7th; pulse never above 96	Cicatrix quite healthy	'Gazette Hebdomadaire,' 1862, t. ix, pp. 401, 488.
Few adhesions; clamp to short pedicle; operation lasted half-hour	Quinine subcutaneous	D. 3 days	Disease began with feeling of constriction at neck; br. pneum. 3rd day; sepsis.	Uterus dragged by stump; no peritonitis	'Deutsche Klinik,' 1866, Bd. xviii, Fall. 10, S. 38.
Incision 4½ inches. Many adhesions to viscera. Pedicle 1½ inches thick, fastened by lancet pins, passing through middle to wound edges	Morphia subcutaneous, gr. ½	D. 7 days	Disease began left maxilla; stump not separated; patient convalescent	Stump adherent to cicatrix; two collections pus in stump; ileum perforated	'Deutsche Klinik,' 1867, Bd. xix, Fall. 11, S. 97, 112, 144.
Navel to pubes incision; pedicle ligatured in two and united to wound, as in case vii. Metallic "constricteur"	Pneumonia treated, trismus not	D. 36 hrs.	8th day severe fright; normal up to onset of attack	Stump loose; firm cicatrix; omentum firmly adherent	'Deutsche Klinik,' 1867, Bd. xix, Fall. 12, S. 204, 213, 227.
4-inch incision. Wire ecraseur round finger-thick pedicle; wire sutures; glass tube beside pedicle; removed 2nd day. Ferri perchlor. to stump	Curari suggested, but not procurable	D. 16 hrs.	Intense peritonitis 3rd day; ecraseur and stump slipped into cavity 3rd day; attack began with stiffness in neck and jaws	Peritonitis	'Deutsche Klinik,' 1868, Bd. xx, Fall. 16, S. 181–4.
7-inch incision. Omentum ligatured *en masse* and brought into wound. Pedicle short, brought through to vagina	Potass. Bromid.; chloroform	D. 10 days	Putrid discharge from vagina 2nd day; attack began with swollen tongue; stiff jaws	—	'Boston Med. and Surgical Journ.,' 1874, vol. xc, p. 521.
8–9-inch incision; right pedicle thick and vascular, left smaller; right burnt through, and ligatured in two places; ditto left. Stumps attached to wound edges	Morphia subcutaneous; solutio curari subcutaneous, gr. $\frac{1}{10}$, $\frac{1}{10}$, $\frac{1}{2}$ into neck and near wound	D. 60 hrs.	Was exposed to draught 6th day; right stump cut through 4th; slight redness of fauces left side	Firm cicatrix; small piece of omentum adherent lower angle of wound	'Deutsche Klinik,' 1869, Bd. xxi, Fall. 19, S. 241–3.
No adhesions. Pedicle ligatured, clamp applied. Five wire sutures	?	D. ?	No blood or ovarian fluid escaped into abdominal cavity	Negative	'Deutsche medic. Wochenschrift,' 1880 Jahrg. vi, S. 50.

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Case No.	Operator.	Hospital or Private House.	Date and Locality of Operation.	Age. M. S. W.	Ch.	Previous History.	Nature of Tumour.	Date of Onset and Termination of Tetanus.
xiii	B. Stilling	P. H.	25, iv, 1872. Helsa, near Cassel	50 ?	0	Rapidly growing tumour; right side 1 year; 7 pints fluid drawn off 8 days previous to operation for rapid emaciation and dyspnœa	Right multilocular cyst of ovary	5th–6th
xiv	B. Stilling	H.	29, v, 1872. Klinik, Cassel	56 w.	11	Tumour in right side 18 months	Multilocular ovarian cyst, right ovary; colloid degeneration; weight 50–60 lbs.	16th–42nd
xv	Tod Helmuth	P. H.	19, x, 1872. Brooklyn, U.S.A.	38 ?	?	Much pain in right side	Multilocular cyst, right ovary	12th–13th
xvi	C. W. F. Uhde	H.	19, xi, 1872. Braunschweig, Germany	37 m.	2	Ascites (?)	Cystoma, left ovary	? –7th
xvii	R. Kaltenbach	H.	? viii, 1873. Freiburg	35 m.	2	Tumour observed during last pregnancy	Small unilocular ovarian cyst	16th–24th
xviii	R. Kaltenbach	H.	? viii, 1873. Freiburg	38 m.	4	Severe abdominal pain	Multilocular ovarian cyst; papillary condition of cyst wall	7th–8th
xix	E. H. Gregory	?	? x, 1874. ? U.S.A.	26 s.	0	?	Multilocular ovarian cyst	4th–5th
xx	Schrœder	H.	? ? 1874. Gynækolog. Klin., Erlangen	?	?	?	Ovarian cystoma	9th–13th
xxi	Schrœder	H.	? ? 1874. Gynæk. Klinik, Erlangen	?	?	?	Ovarian cystoma	? –15th
xxii	Howitz	H.	? ? 1874. Copenhagen	47 m.	?	Swelling in abdomen 2 years	Right ovarian cyst; solid tumour left ovary	7th–10th
xxiii	Gilman Kimball	?	? vi, 1877. U.S.A.	47 m.	?	?	Multilocular ovarian cyst	3rd–5th
xxiv	Theophilus Parvin	P. H.	5, v, 1877. Seymour, U.S.A.	36 m.	8	Tumour 5 years, and with 3 pregnancies; tapped a month before operation	Multilocular ovarian cyst; weight 40 lbs.	5th–6th
xxv	Use of operator's name not permitted	H.	31, v, 1878. Frauenklinik, Leipzig	? ?	?	Two ovarian cystomata; repeated peritonitis	Cystomata of both ovaries	? –14th
xxvi	Zweifel	H.	? xi, 1878. Frauenklinik, Erlangen	40 m.	?	Hard abdominal swelling; diagnostic tapping	Dermoid ovarian cyst	6th–7th
xxvii	K. Slawjansky	H.	7, xi, 1878. Klinik of Medical Academy, St. Petersburg	26 m.	1	Tumour noticed 6 months	Proliferating glandular cystoma; larger than fœtal head	3rd–5th
xxviii	Zweifel	H.	? iv, 1879. Frauenklinik, Erlangen	27 s.	0	Tumour noticed 1 year; diagnostic tapping	Large unilocular right ovarian cyst	10th–13th

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Operation, Treatment of Pedicle, &c.	Treatment.	Result and Duration of Disease.	Remarks.	Post Mortem.	Reference.
5–6-inch incision; many adhesions. Short pedicle, tied with hemp and dropped into peritoneal cavity. No tube; seven button sutures	Chloral Hydrat. per rectum	D. 21 hrs.	Trismus, never tetanus, beginning with dysphagia; septic peritonitis	—	'Deutsche Klinik,' 1872, Bd. xxiv, Fall. 25, S. 396–7.
16-cm. incision. Five catgut ligatures to pedicle; dropped in	Quiniæ Sulph.	R.	Trismus; tetanus of abdominal muscles and diaphragm; albumen and sugar	—	'Deutsche Klinik,' 1872, Bd. xxiv, Fall. 27, S. 418, &c.
Dense adhesions to omentum, liver, intestines, spleen. Ecraseur round whole mass	None special	D. 24 hrs.	Intensely cold weather; fire allowed to go out	—	'Homœopathic Journ. Obstet.,' 1884, p. 14, Case IV.
No adhesions; long pedicle, clamped after ligature. Liq. ferri to stump. Six silver wires	?	D.	Listerism in all details; occupied different ward from Case X; no tetanus between 1869 and 1872 in Klinik	Negative	'Deutsche medic. Wochenschrift,' 1880, Jahrg. vi, S. 50.
No adhesions; clamp; thin pedicle	Chloral per rectum; morphia, chloral, chloroform	D. 8 days	Wound quite healed; non-febrile course to 4th day; last day 42·7° C.	Negative; no peritonitis	Priv. comm. direct from operator.
Many adhesions; divided by cautery; broad, thick pedicle. Clamp	Morphia, chloral, chloroform	D. 24 hrs.	Non-febrile course after 4th day	Negative; no peritonitis	Priv. comm. direct from operator.
Adhesions numerous; intra-peritoneal treatment of stump. Silver wire to incision	None special	D. 36 hrs.	?	—	Parvin's list, 'Amer. Gyn. Trans.,' vol. ii, 1877, p. 317.
Extensive adhesions between tumour and anterior abdominal wall. Clamp	?	D.	?	No peritonitis; wound healed	'Sitzber. der Med. Soc. zu Erlangen,' 1875, Hft. vii, S. 82.
Firm but not extensive adhesions; large amount oozing. Clamp; no drain-tube	?	D.	Fever first day; normal 8 days; no sepsis (?)	Fibrinous flakes on intestines; pus in various cavities	'Sitzber. der Med. Soc. zu Erlangen,' 1875, Hft. ii, S. 83.
Enucleation of tumour; short, broad pedicle. Clamp; left side untouched	?	D.	All symptoms normal up to 7th day	—	Virchow and Hirsch, 'Jahresbericht der Med.' for 1875, ii, S. 570.
Adhesion to colon. Clamp	Chloral per rectum; morphine	D. 48 hrs.	—	—	Parvin's list.
Considerable omental and parietal adhesions; long and thin pedicle. Clamp	Opium, morphine, chloroform	D. 36 hrs.	Difficulty in producing anæsthesia; attack began with pain in neck; extreme cold; open windows.	—	'Amer. Gynæc. Trans.,' vol. ii, 1877, p. 317.
Firm adhesions to uterus; ovaries removed by "écrasement," and stumps dropped in. Wire and short sutures	?	D.	Trismus was of short duration	No sepsis	Private comm. from Prof. Carl Hennig.
Carbolic spray; pedicle pierced and tied with double thread; intra-peritoneal; sutures carbolised	Morphia	D. 24 hrs.	One half of pedicle cut into by ligature; slight fever during convalescence	No peritonitis; omentum much stretched	'Berlin. klin. Wochen.,' 1881, Jahrg. xxviii, S. 307, and private comm.
Silk ligature; stump fastened into abdominal wound	Hydrate of chloral per rectum	R.	Trismus well marked; tetanus very slight	—	Private comm. direct from operator.
Omental adhesions; pedicle pierced and tied with silk; intra-peritoneal	Curari, ·015 grm. and morphia; finally chloroform	D.	Disease began in neck and back muscles; a febrile course up to 10th	Omentum tensely stretched; stump healthy; no sepsis	'Berlin. klin. Wochen.,' 1881, Jahrg. xviii, S. 397, and private comm.

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Case No.	Operator.	Hospital or Private House.	Date and Locality of Operation.	Age. M. S. W.	Ch.	Previous History.	Nature of Tumour.	Date of Onset and Termination of Tetanus.
xxix	Granville Bantock	H.	28, xi, 1879. Samaritan Hospital, London	24 s.	1	*Vide* 'Med.-Chir. Trans.,' vol. lxiv, p. 130; chart	Unilocular parovarian cyst, right ovary; weight 3 lbs.	7th–8th
xxx	Thomas Underhill	H.	17, iii, 1880. Guest Hospital, Dudley	? 40	?	Tumour 7 years	Large right unilocular cyst; 560 oz. fluid. Small left ovarian cyst	6th–7th
xxxi	Werth	H.	23, iii, 1880. Frauenklinik, Kiel	?	?	?	Large unilocular cyst, right ovary	6th–8th
xxxii	R. Olshausen	H.	27, iv, 1880. Frauenklinik, Halle a/S	54 m.	?	?	Proliferating ovarian cystoma; 4½ lbs. weight	11th–13th
xxxiii	Thomas Underhill	H.	22, vi, 1880. Guest Hospital, Dudley	21 s.	0	Intense dysmenorrhœa; unable to follow occupation	Right ovary prolapsed and tender	5th–7th
xxxiv	Salmon	P. H.	12, xi, 1880. Yermenonville, near Chartres, France	26 m.	?	?	Multilocular ovarian cyst	6th–7th
xxxv	Thomas Savage	H.	14, xii, 1880. Hospital for Women, Birmingham, England	51 m.	11	?	Large right cystoma	7th–9th
xxxvi	E. Malins	P. H.	1, iii, 1881. Birmingham, England	60 s.	0	Increasing tumour; under observation 18 mos.	Multilocular ovarian cyst	8th–10th
xxxvii	Knowsley Thornton	P. H.	? iii, 1881. ? England	53 s.	0	Cyst ruptured, and tapping previously; followed by "slight threatening of tetanus"	Semi-solid left ovarian cyst; weight 25 lbs.	? –6th
xxxviii	R. Olshausen	H.	3, viii, 1881. Frauenklinik, Halle a/S	27 m.	?	?	Small proliferating cystoma	17th–19th
xxxix	James M. Bennett	H.	10, viii, 1881. Liverpool	41 ?	?	Tumour 8 years; three tappings previous to operation	Left multilocular ovarian cyst, containing 59 pints. Right ovary cystic	15th–18th

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Operation, Treatment of Pedicle, &c.	Treatment.	Result and Duration of Disease.	Remarks.	Post Mortem.	Reference.
Twisted pedicle; single ligature; intra-peritoneal; pelvic adhesions	Chloroform; chloral, gr. 60; Tinct. Cannab. Ind., 3ij by enema; Ext. Physostig. fab., gr. ¾ hypoderm.	D.	4th day after operation removed to another ward; placed in strong draught	Stump undergoing normal changes; ligature tight; proximal and distal bulging	'Trans. Path. Soc., 1880, vol. xxxi, p. 7, and private comm.
Extensive adhesions to back and fundus of uterus; pedicle tied with catgut. Wire to incision	?	D. 28 hrs.	Wound healed 5th day; attack began with nausea and twitchings	Wound healthy; no peritonitis	'British Med. Journ.,' 1881, i, p. 807, and private comm.
No adhesions; catgut to long pedicle in two parts; intra-peritoneal	?	D.	Trismus first appeared in masseters	No sepsis	'Register Kiel Journ.,' 9598, 49, private comm.
No adhesions; pedicle screwed up with wire tightener; ligatured in two halves with silk; intra-peritoneal	?	D.	?	Negative	'Klin. Beiträge zur Gynæk. und Geburt.' 1884; Stuttgart, "Ovariotomien Tabelle," Fall. 90, and private comm.
3-inch incision. No adhesions; catgut to pedicle; intra-peritoneal	?	D. 40 hrs.	?	No peritonitis; wound healed; wandering leucocytes in cervical cord	'British Med. Journ.,' 1881, i, p. 808, and private comm.
Operation two days before menstruation; no adhesions; pedicle tied in two, then *en masse* and dropped in. Two harelip pins in abdomen; wound. Seven superficial sutures	?	D. 24 hrs.	Pins removed; wound quite healed; spray and Listerian precautions	No sepsis	Maunowry, in Thiriar's paper, p. 102 (*vide* Case XL).
3-inch incision. Pedicle ligatured; dropped into cavity	?	D.	No septic cases or cases of tetanus in operator's practice at time	No peritonitis; pedicle adherent to small intestine and sigmoid flexure; no changes in cord	Private comm. direct from operator.
No adhesions; broad pedicle, tied in three divisions; silk; stitches removed 6th day	Chloral, gr. xx, and morphia, gr. ½ repeated	D. 48 hrs.	Three other cases in same locality in three months	—	'British Med. Journ.,' 1881, i, p. 509.
Omental and intestinal adhesions; ligatures to pedicle	None special	D.	Cold windy weather; violent gust of wind first started spasms	—	'Med.-Chirurg. Trans.,' 1887, vol. lxx, p. 53, and chart, Case 231.
Intimate adhesions to abdominal wall and intestines. Torsion, then ligature of stump, with silk in two portions; intra-peritoneal	?	D.	?	In stump, an abscess, size of a nut, with sweet pus	'Klin. Beiträge zur Gyn. und Geburt.,' 1884, Stuttgart, Fall. 136.
Both pedicles ligatured by transfixion, and dropped into abdomen	Opium and quinine; Atrop. Sulph., gr. $\frac{1}{75}$ subcutaneous (3 doses); Extr. Physostig. fab., gr. ¼ (6); chloroform mixture, gr. ij	D. 72 hrs.	Attack ushered in with shivering; slight sore throat; erythematous blush over arms and legs; temp. 107·2°	Firm cicatrix	'Lancet,' 1881, vol. ii, p. 947.

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Case No.	Operator.	Hospital or Private House.	Date and Locality of Operation.	Age M. S. W.	Ch.	Previous History.	Nature of Tumour.	Date of Onset and Termination of Tetanus.
xl	J. Thiriar	H.	13, vii, 1883. L'Hôpital St. Jean, Bruxelles	18 s.	0	—	Small unilocular ovarian cyst	6th–8th
xli	Thomas Savage	P. H.	24, v, 1884. Warwickshire	34 m.	?	—	Right ovary cystic. Bleeding myoma, size of cricket ball	6th–8th
xlii	G. Consentino	H.	1, vi, 1884. Ospedale Civico, Palermo, Sicily	50 m.	5	Cessation of menstruation three years before from fright. Much pain. Tumour noticed soon after. One tapping	Multilocular cyst of left ovary, size of eight months' pregnancy	5th–7th
xliii	J. Thiriar	P.	17, xii, 1885. Brussels	45 ?	?	—	—	6th–7th
xliv	E. Börner	P. H.	? ? 1886. Gratz, Upper Styria, Austria	50 m.	?	?	Intra-ligamentous ovarian cysts	8th–9th
xlv	J. Thiriar	H.	29, vi, 1886. L'Hôpital St. Jean, Bruxelles	23 s.	0	?	Unilocular ovarian cyst	7th–8th
xlvi	Thomas Savage	H.	3, vii, 1886. Staffordshire	29 s.	0	?	Large left ovarian cystoma	17th–23rd
xlvii	J. Thiriar	P. H.	31, vii, 1886. Nivelles, Brabant, Belgium	45 s.	0	?	Multilocular ovarian cyst	6th
xlviii	Schatz	H.	7, x, 1886. Frauenklinik, Rostock, Germany	34 m.	3	Repeated attacks of hæmatocele	Hæmatosalpinx and hæmatocele	14th–18th
xlix	F. H. Champneys	H.	9, xii, 1886. St. George's Hospital, London	30 s.	0	In 1881. Enucleation two fibroids; two smaller by ligature. Present tumour noticed 2 years	Nodular fibroid tumour of uterus	4th–5th
l	P. Müller	P. H.	23, vii, 1887. Berne	46 s.	0	Catamenia more copious 4 years. Metrorrhagia 2 years. Violent colic, strangury, and constipation	Intra-mural fibroid, size of fœtal head	8th–9th
li	J. Kötschau	Private Ward	2, ix, 1887. Köln a/Rh., Germany	50 w.	5	Menorrhagia 5 years; acute pain 18 mos.; tumour increasing; cystitis	Fibroid size of fœtal head	4th–5th
lii	Thomas Savage	P. H.	4, i, 1888. Birmingham	32 m.	0	Repeated attacks of ovaritis	Small myoma of fundus, with chronic ovaritis and tender ovaries	8th–11th
liii	Carl Hennig	P. H.	13, viii, 1888. Privat Klinik, Leipzig	34 m.	4	Vomiting 6 mos. before. Menstrual irregularity	Two flattened colloids left ovary, incarcerated in cleft broad ligament	Immediately on closure of wound

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Operation, Treatment of Pedicle, &c.	Treatment.	Result and Duration of Disease.	Remarks.	Post Mortem.	Reference.
Operation easy; no complications; intra-peritoneal	Chloral and opium; chloroform.	D. 36 hrs.	On 6th day at 10 a.m. exposed to a strong cold draught; stiffness of neck and jaws 4 p.m.	—	'Comptes Rendus du Congrès français de Chirurgie,' Paris, 2nd Session, 1886, p. 97, Obs. 1.
Short incision; left ovary small and shrivelled; both removed; intra-peritoneal	Chloral, Potass. Brom.	D.	No cases of sepsis or tetanus at time in the practice of operator	—	Private comm. direct from operator.
Firm adhesions to uterus, bladder, and floor; excision of cyst wall; edges sewn to wound. Three drain-tubes	Chloral, morphia; hot baths	D. 36 hrs.	Sutures removed 5th day; febrile course up to 3rd day; temp. then 103°; normal after	—	'Annali di Ostetricia Gin. e Ped.,' 1885, p. 389.
Ovaries removed; no difficulties; operation twenty-five minutes; intra-peritoneal	Morphia, chloral	D. 12 hrs.	Normal progress up to evening of 6th day	?	'Comptes Rendus, &c.' (Case XL), p. 98, Obs. 2.
Partial removal; clamp; no adhesions; total enucleation impossible	?	D. 24 hrs.	Normal convalescence up to 7th day	—	Private comm. direct from operator.
No adhesions; operation fifteen minutes; intra-peritoneal	Cocaine repeated frequently subcutaneously, and combined with morphia	D. 18 hrs.	Attack began with dyspnœa; great relief from cocaine	—	*Vide* Case XL, pp. 98, 99, Obs. 3.
Short incision; no adhesions; ligature to pedicle	?	D.	Left hospital 15th day well; died within week; no sepsis or tetanus in operator's practice	—	Private comm. direct from operator.
No adhesions; operation thirty minutes; intra-peritoneal	Pilocarpine injected shortly after breathing became embarrassed	D. 7 hrs.	Condition up to 6th day good	—	*Vide* Case XL, p. 99, Obs. 4.
Many adhesions; both tubes and ovaries removed; silk ligature to pedicle	Chloral and Pot. Brom.	D.	Phlegmasia after first child	—	Private comm. direct from operator.
Many adhesions; incision to left of old one; ovaries indistinguishable; abdomen closed; difficulty with sutures from muscular irritability	Morphia and Ext. Physostigm. fab. hypod.	D. about 30 hrs.	When wound was dressed shortly before death the whole central part sloughed; (?) tension; full antiseptics and spray	Organs normal; kidneys healthy, but congested	Private comm. and Hosp. Register, P. M. No. 382.
Ovaries removed; no adhesions. Ovaries normal; intra-peritoneal	Morphia as required	D. 20 hrs.	Three bad attacks; the trismus and opisthotonos most marked left side	—	Private comm. from operator's private assistant (Dr. Krumberg).
Healthy ovaries removed; silk ligatures to pedicles; stumps sprinkled with iodoform; intra-peritoneal; Listerism	Potass. Bromid.; chloroform narcosis	D. 24 hrs.	Hallucinations 4th day; trismus same evening	No peritonitis; stumps normal; visceral petechial hæmorrhages	Münchener med. Wochenschr., 1887, xxxiv, s. 855–6.
Appendages very adherent; ovaries contained small cysts; drainage-tube	Eserine	D.	Operation very difficult. No other cases of tetanus at same time	—	Private comm. direct from operator
Many adhesions; tumours gangrenous, slightly torn. India-rubber tube round base; pedicle pierced, tied. Intra-peritoneal	Oil of camphor subcutaneously	D. 6 hrs.	Trismus appeared immediately. Fæcal vomiting	—	Private comm. direct from operator

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Case No.	Operator.	Hospital or Private House.	Date and Locality of Operation.	Age. M. S. W.	Ch.	Previous History.	Nature of Tumour.	Date of Onset and Termination of Tetanus.
liv	Nieberding	H.	13, xii, 1888. Riedinger's Klinik, Würzburg	? s.	?	?	Generally adherent. Right ovarian cyst. Retroverted uterus and small fibroid	9th–13th
lv	P. Horrocks	H.	4, xi, 1890. Guy's Hospital, London	46 m.	7	Increase in size 3–4 yrs. Much left-sided pain lately. Menorrhagia	Ovarian cyst, containing cartilaginous material in cavity	7th–9th
lvi	Thomas Savage	P. H.	9, xi, 1890. Leamington	46 w.	11	?	Large left ovarian cystoma	5th–7th
lvii	John Phillips	Private Ward	21, i, 1891. Lewisham	35 m.	2	Previous operation 7, i, 1889, for (?) malignant growth and ascites; much improved	Large multilocular papillomatous cystoma right ovary. Ascites	5th–6th

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Operation, Treatment of Pedicle, &c.	Treatment.	Result and Duration of Disease.	Remarks.	Post Mortem.	Reference.
Many adhesions. Rupture of cyst, but no escape of contents; right pedicle tied with silk; intra-peritoneal	Chloral (large doses) no effect. Physostigmine, with good effect at first, then cardiac failure	D.	Sutures removed 8th day; normal convalescence; tetanus followed trismus	Negative	Schmid. Inaug. Diss. 'Ueber Ovariotomie,' S. 10, 11, 16, 17, and private comm.
Omentum generally adherent over tumour; piece of omentum ligatured into pedicle; intra-peritoneal	Morphia, gr. $\frac{1}{8}$ every two hours	D. 36 hrs.	No case of tetanus in wards; attack began by numbness at occiput	Wound healed. Stump and viscera quite healthy	Private comm. direct from operator
3-inch incision; some ascites; no adhesions; ligature close to uterus; intra-peritoneal drainage-tube	Extr. physostigmat.; eserine	D.	Wound quite healed. Temperature, pulse, and general condition normal up to 5th	Distal end of pedicle gangrenous	Private comm. direct from operator
Many adhesions; stump very thick, pierced, and tied in four places, carbolic silk; intra-peritoneal. Drain-tube	Chloral; Potass. Bromid. chloroform. Extr. Physostig. fab. gr. $\frac{1}{3}$ three times subcutaneously	D. 36 hrs.	Pulse, temperature, and general condition normal up to onset of tetanus. Attack commenced with slight sore throat and stiffness left side	Wound firmly healed; stump healthy. No peritonitis; no sepsis	Present communication

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ADDENDUM TO TABLE A.

A List of Seven Cases in which full particulars as above could not be obtained.

Boinet.[1]—3 cases in a series of 77 ovariotomies.

Case lviii (2, x, 1865).—Æt. 41. Multilocular ovarian cyst; strong adhesions; clamp; death from tetanus 6th day. (Case 9 in series.)

„ lix (21, ix, 1873).—Æt. 42. Multilocular ovarian cyst; many adhesions; clamp. (Case 52 in series.)

„ lx (8, x, 1874).—Æt. 30. Performed in Paris; numerous adhesions; clamp and ligature; multilocular ovarian cyst. (Case 64 in series.)

J. Boye.[2]—3 cases in a series of 23 ovariotomies (1876–78).

Case lxi.—Æt. 52. No adhesions or hæmorrhage; multilocular cyst, with medium long pedicle; clamp; no drain-tube; died of tetanus on 8th day. (Case 2 in series.)

„ lxii.—Æt. 26. Adhesions. Cyst with purulent contents; long pedicle; catgut ligature, intra-peritoneal; drain-tube in lower angle of wound: died of tetanus 15th day. (Case 9 in series.)

„ lxiii.—Æt. 30. Simple cyst with serous contents; long, thick pedicle; clamp; died of tetanus 9th day. (Case 12 in series.)

Meadows.[3]—

Case lxiv (1884).—Æt. 49. Cyst removed without difficulty, intra-peritoneal; for 5 days kept on ice and water diet; wound healed; became restless; died on 8th day.

In 64 cases there were 61 deaths and 3 recoveries, or a mortality of 95·31 per cent.

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[1] 'Maladies des Ovaries,' 1877, 2nd edition, table, p. 577.

[2] 'Gynækologiske og Obstet. Meddelelser,' Andet Bind, 1879, p. 1.

[3] Private communication from Sir George Humphry.

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TABLE B.

Cases in which Trismus occurred during *operation.*

Operator.	Date and locality.	Age.	M. S. W.	Nature of tumour.	Operation, adhesions, &c.	Result.	Remarks.	Reference.
A. J. Krassowski	Feb. 22, 1865. Frauen. Spital der Med. Chir. Acad., St. Petersburg	35	S.	Multilocular colloid cyst of left ovary	Incision 20 c.m. Pedicle short and fibrous, ligatured in two with silk; intraperitoneal	Died 3rd day; hæmorrhage	1 attack, trismus during operation	St. Petersburg Med. Wochensh., 1884, s. 142-3, F. 3
,,	Aug. 27, 1868. Frauen. Spital der Med. Chir. Acad., St. Petersburg	35	M.	Left ovary cystic colloid; right ovary cystic	Incision 22 c.m. Pedicle fairly long, thin, and not vascular; burnt through; deep peritoneal sutures and superficial metal sutures	Died 4th day; entero-peritonitis	2 attacks, trismus during operation	St. Petersburg Med. Wochensh., 1884, s. 191, F. 35.

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TABLE C.

A Classification of the Possible Causes of Tetanus in the 64 Cases.

1. Nerve-irritation, produced by—
   - The clamp.
   - Over-tight or irregular ligature.
   - Tension of stitches in abdominal wound, whether silk, wire, or button sutures.
   - Stretching, tearing, or ligaturing omentum.
   - "Torsion" treatment of pedicle.
   - Tearing through old and firm adhesions during operation.
   - Cicatricial contraction (more especially in connection with chronic tetanus).
2. Contagion—
   - Direct from another case of tetanus.
   - By "garden-mould" bacillus.
3. Sepsis.
4. Epidemic.
5. Endemic.
6. Miscellaneous—
   - "Tendency" to disease.
   - Predisposing nervous condition of patient.
   - Sudden nervous shock, *i. e.* noise, bad news.
   - Cold draught playing on to patient.
   - Alternating extremes of cold and hot weather.
   - Low diet after operation.

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TABLE D.

Summary of Treatment of Pedicle in 64 Cases.

I. *Extra-peritoneal:*

	Cases.
(1) Clamp to stump in Cases ii, iii, v, vi, xii, xvi, xvii, xx, xxi, xxii, xxiii, xxiv, xliv	= 13
(2) Stump fastened to wound edges in Cases i, vii, viii, xxvii	= 4
(3) Stump fastened to wound edges with application of cautery in Case xi	= 1
(4) Cyst-walls sewn to edges of wound in Case xlii . .	= 1
(5) Wire écraseur to stump in Cases ix, xv . . .	= 2
(6) Stump brought down into vagina in Case x . .	= 1
(7) Combination of clamp and fastening omentum to wound in Case iv	= 1
(8) Combination of clamp with cautery to stump in Case xviii	= 1
	24
Boinet's 3 cases, lviii, lix, lx Boye's 2 cases, lxi, lxiii }	5
	29

II. *Intra-peritoneal:*

(1) Pedicle ligatured and dropped into peritoneal cavity .	= 29
(2) Écrasement of pedicle and then dropped into peritoneal cavity, Cases xxv, xxxii, xxxviii . . .	= 3
(3) Exploratory incision in Case xlix . . .	= 1
	33
Boye's Case, lxii Meadows' Case, lxiv }	= 2
	35

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TABLE E.

Indicating the Proportion of Ovariotomies to Cases of Tetanus occurring among European Operators.

	Total Ovariotomies.	Cases of Tetanus.
Ahlfeld (Marburg)	105	0
Börner (Gratz)	200	1
Bergesio (Turin)	38	1
Braun, Gustav (Vienna)	801	0
Conrad (Bern)	15	0
Credé (Leipzig)	30	0
Cuzzi (Pavia)	80	0
Dohrn (Königsberg)	300	0
Dolèris (Paris)	102	0
Droysen (Gottingen)	102	0
Fehling (Basle)	100	0
Fraenkel (Breslau)	125	0
Fritsch (Breslau)	710	0
Gusserow (Berlin)	450	0
Hennig (Leipzig)	30	1
Herff (Halle a/S)	10	0
Hegar (Freiburg)	600	0
Hergott (Nancy)	15	0
Hofmeier (Würzburg)	100	0
Jeannel (Toulouse)	114	0
Kochs (Bonn)	17	0
Kaltenbach (Freiburg)	400	2
Laroyenne (Lyons)	320	0
Löhlein (Giessen)	120	0
Müller, P. (Bern)	210	1
Münster (Königsberg)	48	0
Nieberding (Würzburg)	25	1
Olshausen (Berlin)	950	2
Pernice (Griefswald)	243	0
Rokitansky (Vienna)	129	0
Schauta (Prague)	300	0
Säxinger (Tübingen)	211	0
Schatz (Rostock)	200	1
Slawjansky (Petersburg)	205	1
Tibone (Turin)	30	0
Veit (Bonn)	450	0
Werth (Kiel)	229	1
Winckel (Munich)	500	0
Wyder (Zurich)	60	0
Zweifel (Erlangen)	460	2
	9134	14

The proportion of tetanus cases among ovariotomies in Europe (excluding the British Isles) is 1 in 652·43.

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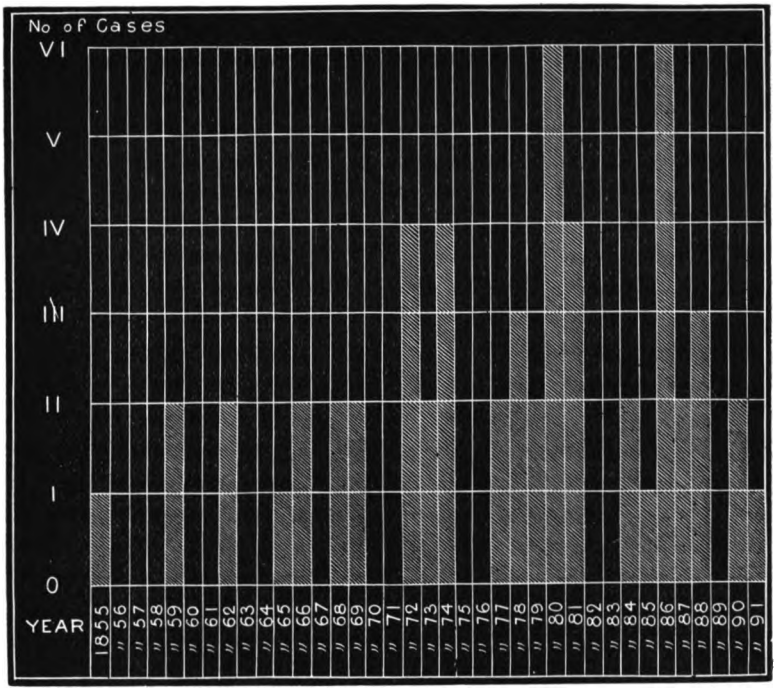
Chart.

Indicating the Number of Cases occurring Annually for Thirty-six years.



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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 47.)

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GLANDULAR SWELLING IN NECK;

CONVERSION INTO A PULSATING TUMOUR LIKE ANEURISM; LIGATURE OF THE COMMON AND EXTERNAL CAROTIDS AND OF THE INTERNAL JUGULAR VEIN; CURE.

BY

JOHN CROFT.

Received October 23rd, 1891—Read January 27th, 1892.

This case appears to be worthy of record, first on account of its pathology; secondly, for the success which attended the treatment; and thirdly, on account of its rarity. If there are precisely similar cases on record, I cannot find them.

A case of abscess in the neck which in its course destroyed a large portion of the common carotid, jugular vein, and pneumogastric nerve is recorded by Sir Wm. Savory in the 'Trans. of the Royal Med. and Chir. Soc.' for 1881, and is somewhat similar. The morbid action in my case may have been of the same quality, but not the same degree. It is to be observed that the case about to be narrated was not one of abscess, if abscess is defined as a collection of *pus* in a cavity. No pus was seen, and no pus was discovered by the operation. Inflammatory exudation of a fluid character there must have been, but burrowing pus was not met with, though doubtless the process by which the vein and artery had been destroyed

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was similar to that by which the same structures are destroyed in the advance of a distinctly suppurative inflammation.

H. H—, æt. 22, bootmaker, was admitted into St. Thomas's Hospital under Mr. Croft's care on June 4th, 1891. He had enjoyed good health until eight weeks before admission. Then he began to complain of sore throat, and a month later he observed a small swelling on the right side of his neck. This swelling gradually increased in size until a week before he applied to the hospital, when it became rapidly larger. Under treatment it appeared to subside somewhat. At the time of his admission the tumour appeared to be about as large as a pullet's egg. It was situated opposite the thyroid cartilage, and was overlapped by the edge of the sterno-mastoid muscle. Its long axis was from above downwards. It was fairly movable. The skin over the most prominent part was slightly adherent, and there a little fluctuation could be felt. It was examined by several persons, but no pulsation was observed by any. It was diagnosed as a glandular abscess which would require incision, and not suitable for excision. No other glandular affection was found, and no tubercular tendency was discovered. During the evening of the second day after admission he complained of fresh pain in the swelling, and suddenly experienced some temporary difficulty in swallowing and breathing. No change was made in the applications. On the morning of the fourth day, June 8th, the House Surgeon inspected the swelling prior to its being operated on; he then found, to his amazement, that the swelling had more than doubled its size, and that it had become strongly pulsatile. The pulsation was carefully examined, and found to be decidedly centrifugal and expansile, and to be accompanied by a slight systolic murmur. No thrill could be felt. The patient's temperature was natural, and he felt fairly well.

On my arrival at the hospital at 1.30 on that day

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(June 8th) my immediate attention was directed to the case, and I found it in the following condition. The tumour, which was the size of a duck's egg, yielded a sense of fluctuation, and pulsated freely with a markedly expansile movement. The integuments over its most prominent part were discoloured, dusky, and adherent from inflammatory exudation. It pushed the sterno-mastoid outwards and extended upwards towards the angle of the jaw, and downwards to within two and a half inches of the clavicle, thus making an oval swelling about three and a half inches in length. The common carotid could be compressed below the swelling so that all pulsation could be stopped; but it was observed that the tumour did not perceptibly diminish, although the pulsation was controlled.

The line of treatment seemed to be distinctly indicated, though there was some difference of opinion with regard to the precise nature of the pulsating tumour. One believed it to be an abscess, and that the carotid artery passed through it. This surgeon attempted to demonstrate this condition by endeavouring to compress the swelling, and the carotid through the swelling. These endeavours were followed by a remarkable result, otherwise I should not stop to mention them; they resulted in a decided diminution in the amount of the pulsation, and in obscuring the expansile character of it. After the manipulation the pulsation was no longer so strong nor so expansile. As a first step in the operation I cut down to the sheath of the vessels, with the object of exposing the common carotid below the omo-hyoid. I purposed to place a ligature loosely round the artery, that the means of controlling hæmorrhage from the interior of the swelling might be absolutely ready at the moment of puncturing the cavity. When I had exposed the sheath of the vessels a colleague came to my side and expressed his confident belief that the swelling consisted of an abscess, and that the pulsation as he felt it was not due to an aneurism. To clear up this doubt, whilst an assistant's finger rested on the partly exposed common carotid

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below, I punctured the still pulsating swelling. Its nature was instantly revealed, for dark clots and blood were sharply ejected into the air. Compression of the trunk below was instantly made from the wound already provided, and no current of blood escaped at this time. The next step was to lay open and explore the cavity of the swelling. This was rapidly done whilst pressure was maintained upon the common carotid. No pus was seen and no collection of serum discovered, but a mass of dark coagulum was turned out. No sac or cyst wall was found, and no opening in any artery was observed. At first no bright-coloured blood escaped, but on turning out the dark clots from the upper part of the collection, bright arterial blood flowed rapidly from that part of the cavity and from above downwards. The tissues which included the source of the bleeding were immediately seized between the thumb and forefinger, and by their means the bleeding was kept controlled, but whether it came from the common artery or the external carotid was at that time uncertain. Attention was next directed to the common carotid trunk below; the sheath was opened and a ligature put on. The upper part of the wound was now investigated for the source of the bleeding. The thumb and finger pressure was relaxed. This was instantly followed by copious venous and smart arterial bleeding; the reapplied finger pressure and forceps quickly controlled this. The cavity was now more deliberately explored. Flake-like fragments were seen in the situation of the internal jugular vein in the deep upper part of the wound, but the structures were difficult to distinguish. The walls were blood-stained and infiltrated with inflammation products, and three or four small but swollen glands clung about the sheath of the vessels near the point of bifurcation. The trunks of the common carotid and internal jugular were not laid bare, and no perforation of the former was noticed. The latter named trunk in the lower part of the wound did not distend or pulsate, and fears were entertained lest air passed or

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might pass down it. As a measure of precaution a silk ligature was placed on this vessel at the same level with the ligature on the common carotid artery. The vein when exposed was empty of blood and clot. The pneumogastric nerve was not seen or disturbed. Again attention was given to the upper part of the cavity. In order to completely expose this and the probable source of bleeding, the wound was extended upwards. When the bleeding from this incision had been arrested, the upper end of the jugular was looked for. It could not be defined. The bleeding was controlled by compressing the structures in the position of the axis of the facial and submaxillary and external jugular veins, and the first part of the external carotid artery. A strong silk ligature was therefore placed around this pedicle. At this stage all bleeding seemed to be under control. From an early period of the operation the patient's breathing had been difficult, and the complexion of his face and neck were very dusky. The tongue had been kept out with forceps. Though the breathing caused great anxiety, the operation was not seriously interrupted by it. The wound was now washed out with mercurial solution, and the cutaneous edges were closed by suture. This process had just been completed when increased difficulties beset the breathing. During a struggle copious fresh venous blood deluged the wound. The upper sutures were rapidly cut, and the wound ripped open. The bleeding was venous only, and came from two sources, one in the side of the wound and the other in the deep upper part. The first was quickly controlled by ligature, but the other was difficult to manage. It was commanded by forcipressure. I believed that the forceps included the upper end of the destroyed vein (jugular). The forceps were left in position, as it was impracticable to put on a ligature above the forceps; moreover the patient's condition as regards respiration, there being frothy pink mucus at the nostrils and congestion of the face and head, was very threatening, and warned us to finish the operation as speedily as possible. The wound

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was again irrigated, a packing of iodoform gauze was made around the forceps, and the antiseptic dressings and bandages were finally adjusted. No more bleeding occurred. In the course of two or three hours the breathing became less laboured, his complexion improved, and the pulse steadied. He quickly rallied after that. The operation was followed by congestion of the left lung, the side opposite to the one operated on. From this he had practically recovered on the eighth day. The superficial dressings were changed twice during the week, but the forceps and packing in the deep part of the wound were not disturbed until the sixth day. On that day the forceps and all but the deepest plug were removed. On the eighth day the last piece of packing was withdrawn. The after progress was very rapid, and the wound had so soundly healed by the twenty-fourth day that the patient was then able to leave the hospital. The ligatures remained buried in the healed wound.

Remarks.—With reference to the morbid action in this rare case, I would venture to observe—

First, that it was a morbid process removed by the operative procedures, and that that fact renders it improbable that it was other than a local disease.

Secondly, that it exerted a more destructive influence over the vein than the artery.

Thirdly, that it was not the result of suppuration, for there was no "pus" seen during the operation, nor any definite collection of fluid inflammation products.

Fourthly, that it was not a septic inflammation, as there was no toxæmia and no pyrexia.

Fifthly, the lymphatic glands which were exposed during the operation were hyperæmic, and in a state of inflammation, but as they were not dissected out, their precise condition was unascertained; nevertheless it may be said almost certainly that they were not tuberculous, nor syphilitic, nor septic.

Sixthly, it is surmised by myself that the deep-lying

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glands were primarily affected by an inflammation; and that the vessels, first the internal jugular vein and later the external carotid artery, were secondarily affected by the inflammatory action; and I surmise further that the ensuing pressure and softening of the coats of the vessels led to their yielding to the force of the blood-current, and so on to the formation of the pulsating swelling.

In conclusion I would direct attention to the following subjects:

1. The diagnosis which presented so much difficulty.
2. The nature of the morbid action which opened and destroyed the vessels.
3. The immediate and ultimate abolition of the inflammatory process and its destructive tendencies, although no collection of pus nor any inflamed glands had been opened or removed.
4. That the external carotid near its origin, and not the common trunk, was the artery involved.
5. The value of the antiseptic mode of treatment in such a case.
6. The use of silk ligatures.
7. The absence of cerebral disturbance after the ligatures of the common carotid and internal jugular.
8. The encouragement given to deal promptly, early, and radically in similar cases.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 59.)

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GELATINIFORM DEGENERATION OF HYDATID CYSTS.

BY

J. MITCHELL BRUCE, M.D., F.R.C.P.,

PHYSICIAN TO CHARING CROSS HOSPITAL AND TO THE HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST, BROMPTON;

AND

A. MARMADUKE SHEILD, M.B., F.R.C.S.,

ASSISTANT SURGEON TO CHARING CROSS HOSPITAL.

Received November 10th, 1891.—Read February 9th, 1892.

THE patient was a man of about forty, by trade a licensed victualler. He first came under the care of Dr. Mitchell Bruce in September, 1890, for "rheumatic" pains in several of his joints, muscles, and nerve-sheaths. At this time a small patch of fine crepitations was discovered in the right mammary region, which was referred to an associated local pleurisy. He was much benefited by a visit to Droitwich, but was seen again some six weeks later for occasional appearances of blood in the sputa, hepatic derangement, and an albuminous, bilious condition of the urine.

In the following May (1891) Mr. Berrill, of South Woodford, discovered that the patient was suffering from

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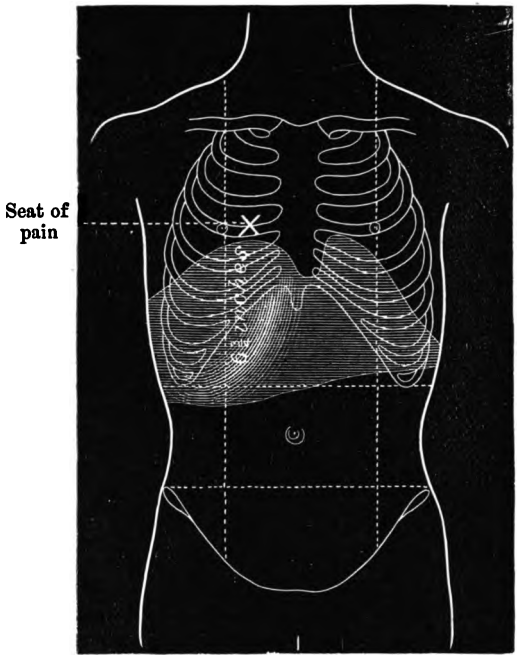
hydatid of the right lobe of the liver, and at a careful investigation of the case on May 12th we determined the physical signs represented on the accompanying diagram (fig. 1). It now became practically certain that the patch

FIG. 1.



of adhesive pleurisy, close above the right wing of the diaphragm, was associated with the hepatic disease, and had been the first evidence of its existence. Pain in the lower part of the right chest and right hypochondrium had become a prominent symptom. It was resolved to watch the case carefully for a month.

On June 9th the physical signs represented in the diagrams (figs. 2 and 3) were elicited. The liver proved to have increased considerably in size within the month. It had risen half an interspace, so that its upper border passed exactly through the right nipple; the percussion

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Fig. 3.

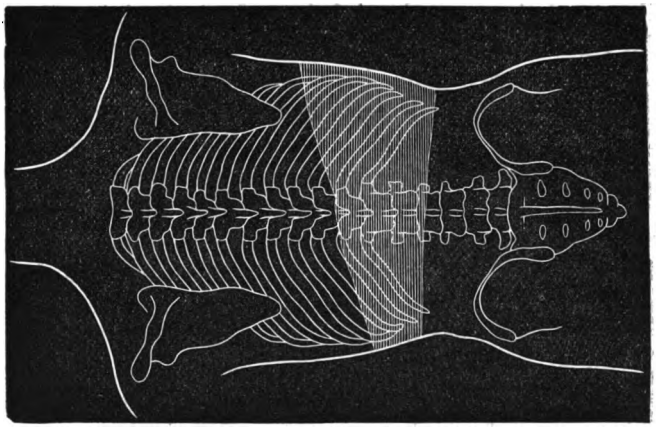
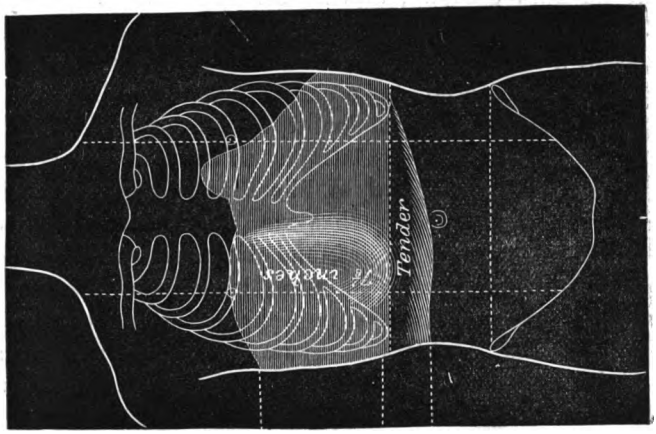


Fig. 2.



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dulness in the right vertical nipple line had increased from 6¾ inches to 7½ inches; and the inferior border was now readily palpable close above the umbilicus. The cartilages of the seventh, eighth, and ninth right ribs, and the right half of the epigastrium bulged markedly, constituting a well-defined considerable tumour, obviously connected with the liver. There was no increase of the pleuritic signs about the mamma, the upper limit of the tumour dulness moving an inch in respiration. Another evidence of the abdominal situation was the outline of the upper border of the dull area, which was arched or convex, as represented in the charts. By this time also the left lobe had considerably increased in size, and the heart was displaced somewhat upwards.

The characteristics of the swelling being those of hydatid cyst, exploratory puncture was decided upon. This was performed on June 23rd. A fine and clean aspirator needle was introduced to a considerable depth, but no fluid escaped, and no sensation was experienced of a cavity having been entered. A stilette was repeatedly passed, and this allowed a few small beads of a tough gelatinous material to escape. On withdrawing the canula, its barrel was found to be blocked with the same material. The gelatinous substance was strongly saline to the taste, and so like that commonly obtained from a colloid cancer, that we were at the time disposed to think that the original diagnosis was erroneous. Mr. Berrill prepared some microscopical specimens of the material, and on inspecting them the next day the characteristic hooklets of hydatids were evidently and certainly present, placing the diagnosis beyond doubt. No ill effects followed the puncture.

As the cyst had progressively enlarged, and was causing severe attacks of pain and great discomfort, it was considered very necessary to remove it; and as its contents were too solid to escape through a canula, abdominal section seemed the only feasible proceeding. The conditions and risks having been explained to the

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patient, he decided upon having the operation performed, and it was carried out on the 30th of June. Dr. Bruce was present, Dr. Frederic Hewitt administered ether, and Mr. Berrill gave valuable and efficient assistance.

The skin of the abdomen, the hands of the operator, and all the instruments were carefully cleaned in carbolic lotion, but no germicides were employed during the operation. An incision was made about four inches long, oblique in direction, from above downwards towards the right, about two inches from the left lower rib cartilages. This incision was planned to expose the more prominent part of the tumour. The tissues, including the greater part of the rectus abdominis muscle, were divided down to the peritoneum without the aid of a director. The abdominal walls were very muscular and vascular, many vessels requiring ligature. When all the bleeding had been arrested, the peritoneum was divided to the extent of the original wound. We had expected to find the cyst wall in close contiguity with the parietal peritoneum, but in this we were disappointed; neither by palpation of the exposed liver was it possible to obtain any evidence of fluctuation. An exploring trocar and canula was now passed into the liver, but it was not until this instrument had penetrated to the depth of three inches that the cyst was reached. The canula was passed in various directions, hoping to reach some fluid, and so reduce the bulk of the contents of the cyst; but nothing escaped save a few beads of the same tough, jelly-like material. The canula being left *in situ* as a guide, sponges were packed all round between the liver and the abdominal wall, and an attempt was made to reach the cyst by cutting through the liver substance with a scalpel. The bleeding was, however, so profuse that had this been persevered in, the patient would have lost much blood, and the peritoneal cavity would certainly not have remained free of clot and serum.

Fortunately the Pacquelin's cautery was at hand; and with the knife blade of this at a dull heat, an incision was

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carried through the liver substance until the cavity of the cyst was reached. The bleeding was even now free, but easily controllable by sponge pressure. Two strong "stay" silk ligatures were passed through the walls of the cyst, the liver, and the abdominal parietes on either side. Owing to the depth of the cyst, and the thickness of the abdominal wall, this was not effected without much difficulty. The liver was by this means brought close to the abdominal wall, and the escape of the contents of the cyst guarded against during evacuation of its cavity. The second stage of the operation, that of emptying the cyst, was now commenced, and this was efficiently done with a common table-spoon, as recommended by Tait. The cyst may truly be said to have been solidified throughout. In the centre there was a tendency towards diffluence, but still there was no distinct accumulation of fluid. The material scooped out by the spoon was gelatinous and firm in consistence like ordinary jelly. It was opaque, of a pale green tint, and intimately mingled with myriads of secondary cysts. These varied in dimensions; some were the size of a small apple, others about the size of grapes, but the majority appeared to be unusually small. Most of them seemed to be in an active state of life, as evidenced by their containing pellucid fluid, and possessing laminated capsules. The cysts and the firm jelly in which they were situated filled a large wash-hand basin to the brim. As much as possible of the laminated membrane of the "mother cyst" was removed, and its extent determined. It was of very large size. Above, it seriously thinned the diaphragm, and encroached upon the thorax; below, it extended to the lumbar vertebræ; behind, it lay in contact with the spine and great vessels. The wall of the cyst was so thin in some places, that much exploration was not deemed advisable. The great cavity was now repeatedly flushed out with a powerful stream of hot water; and lastly, the liver was more intimately attached to the margins of the abdominal wound. The sponges were removed one by one; and well soaked fishing-gut sutures

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were passed through the liver and cyst on the one hand, and the peritoneum and abdominal parietes on the other. The insertion of these was again very difficult, owing to the thickness of the abdominal wall and the depth of the cyst. When the suturing was concluded the liver seemed to be well attached. A large tube was inserted into the cavity, and extensive dressings of iodoform, sal alembroth wool, and wood wool were applied. The operation, from the commencement to the termination of the dressing, lasted one hour and ten minutes. There was no marked shock at its conclusion. A small portion of the gelatinous material was further examined on the same evening. It coagulated firmly with heat and nitric acid, and on being shaken up with distilled water gave a copious precipitate of chlorides with nitrate of silver. Under the microscope it was perfectly amorphous, and "hooklets" were sparsely distributed through its substance.

The after progress of the case may be summarised: The patient rallied well from the operation. The temperature rose on the second day to 101°, and then fell. There was no abdominal pain or tenderness, and the wound did well. About the third day, however, mild delirium came on, and this increased to violence, having all the characters, such as delusions and tremor, associated with the delirium of alcoholism. Finally, the patient refused nourishment; and despite all efforts on the part of Mr. Berrill to sustain his strength, he died on the eighth day. The treatment employed was daily dressing of the wound, washing out the cyst with warm and dilute iodine and water, and the free supporting of the strength by nutrient enemata containing laudanum. The discharge was a little bile-stained and quite free from fœtor, and there was evidence that the cyst had greatly contracted. A complete post-mortem examination was refused, but Mr. Berrill informed us in a note that the cyst and abdominal wound had intimately united, that the wound looked healthy, and that there was no peritonitis. The local conditions were therefore all that one

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could desire, and we are inclined to attribute the unfortunate termination to the previous alcoholic condition of the patient.

Notwithstanding the unfavourable result of the treatment of this case, we have thought right to bring it before the profession, because it is an example of a condition of hydatid cyst obviously fraught with much clinical importance, and because it adds another item to the surgery of an important viscus.

It will be convenient to first draw attention to the subject of solidification of hydatid cysts in general, and to gelatiniform degeneration in particular, mentioning its clinical and pathological aspects. We propose to conclude by shortly referring to the surgical measures adopted in this, and advisable in similar cases.

Doubtless the best known and most common cause of solidification of hydatid cyst is caseation of the contents, followed by calcification and shrinkage. This degenerative process, a kind of spontaneous cure, is treated of by many authors, and is in the experience of all who have been much engaged in practical pathology; we need not further allude to it.

The next variety of solidified hydatid to which we wish to refer has not received the general attention which it deserves. Its clinical symptoms are misleading during life, and even post mortem its true nature is sometimes misunderstood. We refer to that variety of hydatid disease known as "echinococcus multilocularis." A distinct tumour is formed, with a fibrous capsule and a tough stroma. In the interstices of this stroma is a gelatinous material, so that the appearance, to the naked eye, is much like alveolar cancer. Only microscopical examination and minute inspection determine the true nature of the disease. Much discussion has arisen whether the cysts are developed along the lymphatics or the blood-vessels; possibly both conditions occur. Among the writers who have given good accounts of this important variety of hydatid may be especially mentioned

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Niemeyer,[1] Frerichs,[2] and Leuckart.[3] It may be especially pointed out that no fluid would be obtained on aspiration, and that the removal of the tumour would be peculiarly difficult owing to the infiltrating nature of the hydatid growth.[4]

We have ventured to apply the term *gelatiniform degeneration* to the peculiar condition exemplified in the case which we have now related. We may preface our remarks by saying that this degeneration must either be rare, or that it has been disregarded by pathologists. We have carefully looked through the scattered literature of hydatid disease, and, with a few exceptions, we do not find any marked allusion to this condition. Gardner, in his well-known article on the treatment of hydatid disease in Australia, does not mention it, though he tabulates forty-two cases, and his experience is undoubtedly large.[5] Graham, whose experience was also obtained in Australia, does not formally draw attention to the condition. He has, however, obviously seen it, for he states that the contents of dead cysts are sometimes tough, tenacious, and colloidal, so that they will not flow through a canula.[6]

Perhaps there is no more valuable information to be obtained regarding hydatid disease in this country than that found in the Thesis of Dr. Balding.[7] He tabulates all the cases of hydatid published in this country by the leading authors, as Harley and Murchison, and from the principal medical journals for the previous fourteen years. He collects the large number of 258 cases. In 198 of these express particulars are given of the nature of the fluids evacuated. In two instances the contents of the

[1] Niemeyer's 'Practical Medicine,' p. 673.

[2] Frerichs' 'Clinical Treatise on Diseases of the Liver,' vol. ii, p. 271.

[3] 'The Parasites of Man,' by Leuckart. Translated, &c., p. 624.

[4] See also Graham, 'Hydatid Disease in its Clinical Aspects,' p. 63.

[5] 'Glasgow Med. Journal,' 1886, p. 368.

[6] 'Hydatid Disease in its Clinical Aspects,' by James Graham, M.D., p. 48.

[7] 'Hydatid Disease of the Liver, its Diagnosis and Treatment,' by Mortimer Balding, M.D., 1880.

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cyst are said to be gelatinous. We think this good evidence of the infrequency of gelatiniform degeneration. One of the instances above mentioned is obtained from Harley's well-known table in the Society's 'Transactions,'[1] A hydatid tumour burst spontaneously near the umbilicus of a woman, and a pailful of a peculiar gelatinous material escaped. In the second instance Dr. Andrew relates in the 'Lancet' for 1875,[2] that the fluid was glairy and gelatinous. As upwards of an ounce of fluid escaped through the aspirator, the contents of the cyst could scarcely be considered as purely gelatinous in this case.

Dr. James Barrett, of Melbourne, relates a case in the 'Australian Medical Journal'[3] which is of great importance, and to which we will briefly allude. A man aged twenty-seven had a large swelling in the left hypochondrium, which presented all the characteristics of hydatid disease. It had previously been tapped, but nothing had escaped. The swelling was now again tapped, but nothing "came out." A fine stilette of whalebone was passed down the canula. Still no fluid was obtained. The patient died a few days afterwards with rigors, vomiting, pain, and fever. At the autopsy the diagnosis was verified. There were two hydatid cysts: a superior one containing decomposing fluid, and being on the point of bursting; an inferior cyst into which the trocar had penetrated. This lower cyst was full of secondary cysts undergoing gelatinoid degeneration. In an article in the 'Medical Times and Gazette'[4] Dr. Barrett refers to this case. "No one could understand," he says, "why no fluid came away from a distinct cyst, and why the stilette was not even moistened. However, the gelatinoid degeneration explained it." In the same article he refers to another case where the change had just begun.

There seem to be gradations in the process, from fluid

[1] 'Med.-Chir. Trans.,' vol. xlix, p. 144, 1866.
[2] 'Lancet,' p. 799, December 4th, 1875.
[3] 'Australian Med. Journal,' June 15th, 1883.
[4] 'Med. Times and Gazette,' 1883, vol. ii, p. 680.

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which is glairy and colloidal, to complete gelatinous solidity. We regret that a detailed chemical examination was not made of the material in our case. From the characters already related, and especially from its highly albuminous nature, we are disposed to look upon it as produced by a colloidal degeneration of the capsules of the secondary cyst, brought about by their mutual pressure. The faint greenish colour may be due to the admixture of biliary fluids, or of the mucus from the bile-ducts. Looking upon the process as a degeneration, it is possible that death of the entire parasite may ensue, with caseation and subsequent shrinkage. In appearance the substance resembled mucin, but its highly albuminous character negatived the view that it possessed this composition. It is possible, however, that the material may have been formed by mucin from the bile-ducts mingled with albuminous substances from other sources, such as the walls of the secondary cysts.

The case which we have related, and the few similar cases to which we have been able to refer, illustrate the clinical importance of the condition. It is evident that no fluid can be obtained from a cyst of this nature, and hence the diagnosis may be dubious or misunderstood. It is obvious that the treatment of gelatinous cyst is beset with graver risks than is the treatment of the ordinary hydatid containing abundant fluid.

The general operative treatment of hydatid of the liver has been fully discussed in Gardner's article;[1] in the 'Australian Medical Journal;'[2] by Graham;[3] and by Davies Thomas. A very complete account of the operative treatment of echinococcus cysts is given in an article by the last-named in the 'Transactions of the Intercolonial Medical Congress of Australasia.'[4] In all the cases treated

[1] 'Glasgow Med. Journal,' 1886.

[2] 'Australian Med. Journal,' 1888.

[3] 'Hydatid Disease in its Clinical Aspects,' p. 145.

[4] 'Transactions of the Intercolonial Medical Congress of Australasia,' Series ii, p. 352, "The Operative Treatment of Echinococcus Cysts," by Davies Thomas, M.D.; see also an article by Gardner, p. 345.

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by direct hepatotomy it seems to be assumed that the cyst itself was exposed by the incision, and the knife used for opening it. No especial mention is made of the fear or treatment of hæmorrhage. Tait, in the cases published in the 'Birmingham Medical Review,'[1] alludes to division of liver substance. He used the knife in all cases to open the cyst. In one instance, liver tissue from a half to three quarters of an inch thick was divided, but there is no special mention of bleeding. In another case he states: "The thickness of liver substance through which I passed was nearly an inch, and I had some little trouble with hæmorrhage, which was, however, completely controlled by pressure." In 1883 Croft attempted to excise a pyriform hydatid of the liver,[2] and it is stated that the parenchyma of that organ bled so freely that he abandoned the attempt, and had recourse to other measures. Greig Smith, in his well-known work on 'Abdominal Surgery,' makes no great point of the treatment of hæmorrhage in hepatotomy.[3] "Bleeding," he says, "may be arrested by a squeeze with pressure forceps, or if this fail, by a continuous suture of not too fine silk." It is obvious, again, that he is considering those cases of cyst or abscess where the layer of liver tissue between the cyst and the operator is insignificant. Jacobson, in his recent work on the 'Operations of Surgery,' follows the practice of Tait in using the knife, plugging the opening thus made with the finger, and subsequently enlarging it by the same means. Hæmorrhage would be arrested by the pressure of the fingers, or by sponge pressure.[4]

The thermo-cautery was used by Rausohoff in 1882 for opening hepatic abscess.[5] It is stated that about half an inch of liver tissue was divided by this instrument. Treves, in his recent work on 'Operative Surgery,' quotes

[1] 'Birmingham Medical Review,' vol. x, pp. 345—351.
[2] 'Med. Times,' November 24th, 1883, p. 597.
[3] Greig Smith, 'Abdominal Surgery,' p. 584.
[4] Jacobson, 'The Operations of Surgery,' 2nd edit., p. 844.
[5] 'New York Medical Record,' vol. xxii, 1882, p. 260.

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a case when Tarsini dissected out a hydatid cyst of the liver. Bleeding, which was very considerable, was checked by ligature and temporary pressure, the patient recovering.[1]

The point of primary surgical importance in the case related is the question of incising liver substance. Should the cyst be in close proximity to the surface the operation is much simplified, for it may be safely opened with a scalpel. But when some three inches of liver substance have to be divided, it requires considerable hardihood to incise it boldly with a scalpel, with the knowledge that large venous channels may be opened, and that uncontrollable hæmorrhage may ensue. The bleeding seemed to us to closely resemble the hæmorrhage from a wounded kidney; and we cannot but think that exposure, time, and sponge pressure would be equally efficacious in checking bleeding from divided liver as divided renal tissue. A free opening into the cyst is very important. It enables all the contents, and especially the shreds of laminated membrane, to be removed. A very large drainage-tube can also be inserted. This insures the cyst being well washed out, and facilitates the escape of the shreds of membrane which subsequently separate.

This case also illustrates the extreme difficulty of ascertaining the relation of the cyst to the parietes, and shows us that the greater prominence externally does not necessarily correspond to the most superficial part of the cyst internally.

Looking upon gelatiniform hydatid in the light of a degeneration, the surgeon will do well to ascertain whether the cyst is in progressive growth, and causing symptoms grave enough to justify operation, before he advises a proceeding which must be attended with some risk.

We would here enunciate the following propositions:

(1) That gelatiniform hydatid is rare.

(2) That when it occurs the diagnosis is difficult, owing to the non-escape of fluid, but that microscopical examina-

[1] Treves, 'Operative Surgery,' vol. ii, p. 457.

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tion of the material in the barrel of the aspirator will aid an exact diagnosis.

(3) That supposing the conditions justify it, abdominal section and evacuation of the cyst by operation afford the best chance for recovery.

In conclusion, we venture to hope that this case and paper may add somewhat to our knowledge of the clinical diagnosis of hydatid cysts, and also may be a small but not unimportant addition to hepatic surgery.

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THE CHANGES IN THE BLOOD IN THE COURSE OF RHEUMATIC ATTACKS.

BY

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Received November 11th, 1891—Read February 9th, 1892.

PART I.—*The variations in the Number and Worth of the Red Corpuscles; and the absence of any recognisable relation between these changes and the appearance of Uro-hæmatoporphyrin in the Urine.*

THAT attacks of acute rheumatism are attended by profound alterations in the quality of the blood is evidenced by the conspicuous anæmia which is so frequently observed in the course of this malady, and which in some instances persists for a considerable time after convalescence is established.

These changes have been studied by two or three observers, whose accounts, although agreeing in several essential points, exhibit nevertheless some rather remarkable differences.

M. Besnier[1] states that Malassez observed a notable

[1] 'Dict. Encyclop. des Sc. Méd.,' article "Rheumatism," p. 492.

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diminution of red corpuscles in this disease, and quotes some observations which demonstrate a fall, followed by a rapid recovery, in the number of corpuscles. In one case the number was reduced to less than two and a half millions in the cubic millimetre.

In their papers on Clinical Hæmatology[1] Drs. Baxter and Willcocks class rheumatic fever with some other acute diseases in which they were unable to detect any noteworthy changes in the blood; but they only give the results of two observations of the blood of a single patient.

M. Hayem's account[2] is by far the most complete which has been given, and I may perhaps be allowed to quote it in full. He writes as follows:—"Acute articular rheumatism is one of the diseases most destructive to the corpuscles. Since salicylic acid has been employed as an efficacious treatment I have had opportunities of comparing cases of almost equal severity, treated, some with this drug and others with quinine, and I think that I may conclude from them that the degree of anæmia produced by the rheumatism is chiefly determined by the duration of the attack. The shorter the attack, and the more rapidly it is arrested by the salicylic acid, the less pronounced is the anæmia. Yet in really acute cases with multiple articular lesions the diminution of red corpuscles is rarely less than a million. In lingering cases or cases with frequent relapses it reaches one and a half to two millions.

"The blood crisis and the repair follow very exactly the course of the accidents of the attack, or rather of the temperature, and local lesions which persist after the fever has subsided retard but do not entirely prevent the repair.

"One finds, then, in acute articular rheumatism a definite blood crisis of rapid development, as in the simplest cases of pneumonia, or successive and subintrant

[1] 'Lancet,' 1880, vol. i, p. 398.

[2] 'Du Sang,' 1889, p. 916.

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crises, or, again, a protracted crisis analogous to that which follows after continuous fevers.

"The variations in the worth of the corpuscles depend usually upon the form of the crisis. Here, as always, it is at the moment of the appearance of many new and incompletely developed corpuscles that the diminution of worth is greatest; but it is not uncommon to see, especially in young subjects, with perhaps a certain chlorotic tendency, a progressive diminution of worth during a tedious recovery, which brings about a condition of more or less persistent chronic anæmia."

M. Hayem further directs special attention to the increase of white corpuscles in this disease.

The most recent contribution to the subject is that of Drs. Maragliano and Castellino,[2] who, without entering into any detail, give a brief epitome of the results obtained from the examination of the blood of fifteen patients suffering from rheumatic fever.

These observers state that there is a profound alteration of the red corpuscles in the course of rheumatic attacks, which alteration affects both the central and peripheral portions of the corpuscles, and is both chromatic and morphological. They found a diminution in the number of red corpuscles, but failed to detect any actual increase of white corpuscles; the apparent increase being ascribed by them to the relative diminution of red corpuscles. They found that the changes observed were proportional to the duration of the attack, to the height of the fever, and to the general condition of the patient; whilst the restoration of the corpuscles during convalescence was very slow and uncertain. They remark that when the heart is attacked the changes in the blood incident to such complications are superadded to those directly attributable to the rheumatic infection.

[1] The worth of the individual corpuscle is obtained by dividing the percentage of hæmoglobin by the percentage of red corpuscles (5,000,000 = 100 per cent.).

[2] 'Gazzetta degli Ospitali,' 22 Marzo, 1891, No. 23, p. 196.

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Having thus given an epitome of the previous literature of my subject, I may proceed to the discussion of the results of my own observations, and shall confine myself in this part of my paper to the variations in number and worth of the red corpuscles, reserving for a future communication the consideration of the variations in the number of white corpuscles, and certain other phenomena. I may, however, remark that all the evidence which I have hitherto obtained points to the occurrence of a true leucocytosis in this disease, and I am convinced that the statement of the Italian observers upon this point is erroneous.

My conclusions are based upon more than eighty observations of the blood of twenty patients at the West London Hospital, and I may here express my great indebtedness to my colleagues on the staff of that hospital for allowing me every facility for observing the cases under their care.

In a few cases the blood was only examined on one occasion, but in others as many as six or eight examinations were made at intervals varying from two or three days to a week. The blood was in all cases obtained from the lobe of the ear, which I find preferable to the finger for this purpose.

The counting of the corpuscles was performed with a hæmocytometer manufactured by Zeiss after a model suggested by Thoma. This instrument has one important advantage over those usually employed in this country, in that the floor of the cell is divided into much smaller squares, by which the counting is greatly facilitated. The dilution is performed in an instrument after the pattern of Potain's mélangeur.

The graduated chamber has a depth of ·01 of a millimetre, and upon its floor is a square divided into 400 lesser squares, each $\frac{1}{400}$ of a square millimetre in area. Each set of sixteen squares is bounded by a triple line. In each observation referred to in this paper sixteen sets of sixteen squares, or 256 in all, were counted, corre-

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sponding to thirty-two squares of Gowers' hæmocytometer.

I have compared my instrument with two of Gowers' pattern, using in each comparison the same drop of blood, with the following results. It will be seen that my instrument gave somewhat higher readings than either of the other two.

Thoma-Zeiss hæmocytometer .	5,890,625	red corpuscles	in the c.mm.
Gowers' hæmocytometer, No. 1 .	5,753,125	„	„
Difference . .	137,500		
Thoma-Zeiss hæmocytometer .	5,206,250	„	„
Gowers' hæmocytometer, No. 2 .	5,100,000	„	„
Difference . .	106,250		

For the estimation of the hæmoglobin a von Fleischl's hæmometer was employed. I have several times made repeated observations with this instrument, employing fresh specimens of blood from the same patient, and have obtained readings in some cases identical, in others differing by no more than 1 per cent. I believe that this form of hæmoglobinometer reaches as high a degree of accuracy as can be attained to in such instruments. The standard of hæmoglobin is, however, somewhat high, and I have never found any blood to attain to 100 per cent. with my instrument. I have compared the instrument with a Gowers' hæmoglobinometer, and found that on two separate occasions the von Fleischl's instrument gave readings which were 8 per cent. lower. Unquestionably errors will creep into determinations with any form of hæmocytometer or hæmoglobinometer, however carefully the manipulations be performed, and it would undoubtedly be a great mistake to argue anything from isolated results, which may always be incorrect; but I think that the charts appended will show that, as an ordinary rule, the errors do not materially affect the results, since the variations are sufficiently great to exceed their limits. A reference to the charts will show that the lines connecting a series of observations are frequently continuous, and that in

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many instances the worth of the corpuscle remains practically constant in spite of great fluctuations in the percentages of corpuscles and of hæmoglobin. Again, the large number of squares counted in each observation must tend to reduce materially the errors of experiment.

In order to afford a standard of comparison I append the results of the examination of the blood of ten healthy persons, and also of ten chlorotic patients with the same hæmoglobinometer and hæmocytometer which were employed in the examinations of rheumatic blood.

Healthy persons:	No. of red corpuscles per cubic mm.	Per cent. of corpuscles.	Per cent. of hæmoglobin.	Worth.
1. Male, aged 34	5,056,250	101·125	82	·81
2. Male „ 22	5,240,625	104·81	80	·76
3. Male „ 23	6,665,625	133·3	82	·61
4. Female „ 28	5,175,000	103·5	80	·76
5. Male „ 24	4,690,625	93·81	81	·86
6. Male „ 15	4,821,875	96·42	77	·79
7. Female „ 27	5,453,125	109·	66	·60
8. Male „ 23	5,468,750	109·3	90	·82
9. Female „ 21	5,028,125	100·5	72	·71
10. Female „ 40	4,990,625	99·8	86	·86
Chlorotic patients:				
1. Female, aged 19	3,962,500	79·25	45	·56
2. Female „ 21	3,009,375	60·2	39	·64
3. Female „ 19	4,915,625	98·3	37	·37
4. Female „ 18	3,571,875	71·4	35	·49
5. Female „ 21	4,031,250	80·62	26	·32
6. Female „ 17	3,828,120	76·6	26	·34
7. Female „ 16	2,871,875	57·4	20	·34
8. Female „ 18	3,134,375	62·7	22	·32
9. Female „ 24	2,575,000	51·5	20	·38
10. Female „ 23	2,925,000	58·5	22	·37

Variations in the Number of Red Corpuscles during Rheumatic Attacks.

An attack of rheumatism is always attended with a considerable diminution of the number of red corpuscles. The fall commences very early in the attack, and is not

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infrequently already at an end when the patient is admitted to a hospital. In ordinary cases, treated with salicylate, the fall usually comes to an end simultaneously with the defervescence and relief of the articular pains; in other words, with the subsidence of the attack. When the attack is at all acute the loss of corpuscles, as M. Hayem states, is usually about a million; as may be seen from the following table, which gives the maxima and minima of red corpuscles in the cubic millimetre observed in ten cases:

	Maximum.	Minimum.	Difference.
1. Male, aged 19 . .	5,106,250 ...	3,815,610 ...	1,290,640
2. Male „ 16 . .	5,684,375 ...	3,909,300 ...	1,775,075
3. Male „ 20 . .	5,618,750 ...	4,721,875 ...	896,875
4. Female „ 18 . .	4,871,000 ...	3,832,000 ...	1,039,000
5. Male „ 34 . .	4,918,750 ...	3,859,375 ...	1,059,375
6. Male „ 9 . .	5,140,625 ...	4,221,875 ...	918,750
7. Female „ 10 . .	4,675,000 ...	3,906,190 ...	768,810
8. Male „ 17 . .	6,200,000 ...	4,978,125 ...	1,221,875
9. Male „ 18 . .	5,109,000 ...	4,136,000 ...	973,000
10. Female „ 22 . .	5,578,000 ...	5,267,000 ...	311,000

The fall of corpuscles is usually extremely rapid, and I have several records showing the loss of about a million in as short a space of time as four or five days.

When the case runs a favorable course, and no serious visceral troubles supervene, the subsequent recovery of corpuscles is equally rapid, so that within ten or eleven days a million red corpuscles per cubic millimetre may be lost and recovered. For example, a patient (Chart I) was found to have 4,686,000 corpuscles on February 26th, and on March 10th the number was nearly the same, 4,871,000; yet in the interval between these two dates the number had been as low as 3,832,000. Here, as in other instances, the hæmoglobinometer confirmed the observations with the hæmocytometer, and throughout the period referred to the variations of worth obtained were very trifling (·57—·53).

In another instance (Chart III) the red corpuscles

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numbered 4,968,750 on July 2nd, and on July 14th, twelve days later, 5,106,250; yet on July 7th there were only 3,915,625, and on the 10th 3,873,437. Here, again, there was an equivalent fall in the percentage of hæmoglobin. Further instances might be quoted in illustration of the rapidity of the loss and recovery of red corpuscles.

The development of a fresh rheumatic manifestation during convalescence will be attended by a repetition of the fall, and the blood chart may thus show a series of such crises (Charts III and IV).

So far my observations agree entirely with those of M. Hayem, but there are other points in which they differ from his. I have not found that in protracted cases there is a continuous fall of corpuscles, but, on the contrary, that the number of corpuscles having reached a certain level tends to remain practically constant until recovery sets in. It must be borne in mind that although two consecutive countings give nearly the same number, there may nevertheless have been a further fall in the interval, and the observations may cut the falling and rising curves at about the same level. I have, however, charts to which no such explanation can possibly apply.

In one case (Chart II) there were no less than five consecutive countings, extending from April 7th to May 4th, in which the number of red corpuscles did not vary by so much as 216,000. During this period the patient was extremely ill with pericarditis with effusion, followed by pneumonia and effusion into both pleural cavities. In another case (Chart VIII) the numbers obtained on four successive occasions (September 28th to October 8th) only varied between 4,137,500 and 3,906,190.

Another point in which my results differ from those of M. Hayem is in the relation between the blood changes and the temperature curve. Undoubtedly in an ordinary typical attack of rheumatic fever the temperature curve affords a very clear indication of the activity of the morbid process, and when the temperature falls the blood-

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corpuscles recover; but it sometimes happens that the number of corpuscles continues to fall after the temperature has reached the normal. Thus in one case the temperature became normal on October 30th, but between this day and November 2nd the corpuscles fell from 5,584,375 to 4,862,500, and there was a corresponding fall in the percentage of hæmoglobin. Still more convincing evidence that the blood-changes are independent of the temperature is afforded by Charts III and IV. The first patient was a young man whose rheumatic attacks were of the type so often seen in children. He had been in the hospital a year previously with the largest rheumatic nodules I have ever seen. He was readmitted with pericardial friction and slight arthritis of the shoulders. The temperature was normal. He had some subcutaneous nodules on the elbows. The blood-corpuscles were found to number only 3,843,700, and he was obviously anæmic. With the disappearance of the friction-sound the blood rapidly recovered, and over a million corpuscles were regained in nine days. He then developed a copious eruption of erythema marginatum, and again lost a million corpuscles. This fresh loss was attended by a corresponding fall of the hæmoglobin percentage, and was confirmed by a second observation three days later. During the erythematous attack the maximum temperature recorded was 100·6°, and that after the fall of corpuscles had already been observed.

Chart IV illustrates the same point. It is that of a young man who was in the hospital for his third rheumatic attack during the present year. The fall of corpuscles observed on August 28th corresponds to a fresh development of pleuritic friction, and that of September 19th to a return of pain in the ankles. Neither of these events was attended by any febrile disturbance whatever, the temperature never reaching 99°.

It will be gathered from these examples that, far from following the temperature curve, the blood curve affords a far more delicate indication of the activity of the rheu-

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matic process. They show further that the changes in the blood are the same whatever the nature of the local manifestation with which they are associated, whether arthritis, pericarditis, pleurisy, or erythema; and point to a latency of the rheumatic process between the appearances of the successive manifestations in cases of this type.

The question presents itself whether the changes in the blood above described have any connection with the salicylic treatment, since Dr. Noel Paton has shown ('Journal of Anatomy and Physiology,' vol. xx, 1885-6, p. 667) that the salicylates have a hæmolytic action, reducing the number of red blood-corpuscles. The statement of M. Hayem that he has found the changes, alike in cases treated with quinine and with salicylate, at once disposes of this idea, and if any further evidence is required, it is to be found in the charts. In each of these a line is drawn along the top indicating the period during which the salicylate was given, the double line indicating frequent doses. In the case represented by Chart III salicylate was only given on two days, when the erythematous rash was at its fullest development; and this chart shows that the blood-changes may be perfectly well marked apart from this treatment. Again, the patient whose chart is numbered II only took salicylate during the first twelve days of his stay in the hospital.

Moreover the reduction in the number of corpuscles observed by Dr. Noel Paton was considerably less than that which occurs during rheumatic attacks.

In some cases there is a fall of corpuscles when the patient first leaves his bed, but the effect of getting up would seem to vary in different cases; for in some instances there is at this period a fall of hæmoglobin only (Chart I), and it is difficult to be certain whether such discordant results may be rightly attributed to this cause.

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Variations in the Worth of the Red Corpuscles.

In many cases the variations in the percentage of hæmoglobin follow those of the corpuscles, and the worth of the individual corpuscle (obtained by dividing the percentage of hæmoglobin by the percentage of corpuscles) remains practically constant. When this rule is departed from there may be in some instances errors of observation; but it is so common to find variations of hæmoglobin independently of the corpuscles, that I am convinced that such variations cannot be all due to such errors. Indeed, there is, in rheumatic patients, a great tendency to more or less rapid falls of worth, which may occur during the acute stage, and be rapidly recovered from; or they may take place during convalescence, and be continuous unless interrupted by treatment. This latter event is that to which M. Hayem has directed particular attention, and it cannot be doubted that it is to such losses of hæmoglobin that the chronic anæmia which sometimes follows a rheumatic attack is due.

In illustration of this point I may refer to Charts V and VII. In Chart V the worth of the corpuscle fell rapidly whilst the number of corpuscles was increasing, but when the patient was put on iron, on March 10th, a recovery immediately commenced.

The patient whose chart is numbered VII left the hospital with falling hæmoglobin and rising corpuscles, in spite of the administration of iron; and during his sojourn in the hospital the worth of the individual corpuscle fell from ·69 (1 equals normal) to ·59.

Falls of worth during the acute period are illustrated by Charts III, VII, and VIII. They do not always occur at the period when large numbers of new corpuscles are being thrown into the circulation, but may take place while the number of corpuscles remains constant, or when the corpuscles are also falling, the percentage of hæmoglobin falling still more rapidly.

Nevertheless it is not very common, even in children,

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for the patient to leave the hospital with a much lower worth than is obtained at the first examination of the blood, however striking the anæmia may have been during the acute stage.

The following table, which gives the worth at the time of the first and last observations in eleven cases, illustrates this point:

Name and age.	Worth at 1st obs.	At last obs.	Difference.
A. B., 10 . . .	·59 ...	·62 ...	+·03
C. T., 9	·65 ...	·64 ...	−·01
W. B., 18 . . .	·70 ...	·71 ...	+·01
H. G., 16 . . .	·59 ...	·65 ...	+·06
H. R., 20 . . .	·69 ...	·59 ...	−·10
F. H., 18 . . .	·90 ...	·76 ...	−·14
E. P., 18 . . .	·51 ...	·42 ...	−·09
W. F., 17 (1st attack) .	·65 ...	·69 ...	+·04
M. A., 22 . . .	·57 ...	·59 ...	+·02
E. C., 34 . . .	·74 ...	·68 ...	−·06
W. F., 17 (2nd attack) .	·65 ...	·64 ...	−·01

In a considerable number of cases the worth of the corpuscle was low at the time of the first observation, but I have had no opportunity of ascertaining whether there is apt to be a fall of worth during what may be called the incubative stage of the disease.

More remarkable even than the falls of worth during the attack is the rapid rise of hæmoglobin percentage independently of any increase in the number of corpuscles. This is a comparatively rare phenomenon, but I am convinced that it does sometimes occur. In the case of the boy whose chart is numbered II the hæmoglobin rose no less than 18 per cent. between April 21st and May 4th, whereas the percentage of corpuscles remained practically constant. This result was so striking that both observations were immediately repeated *de novo* with a fresh drop of blood, and the results of the second examination agreed closely with those of the first. Similar double observations were made on May 11th, when the worth of the individual corpuscles was found to be once more

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falling rapidly. It should be noted that on May 2nd the patient's urine was bright pink, but limpid; it gave the guaiacum reaction and contained albumen. Such urine was passed on two or three occasions only, and there was pain in micturition when it was passed. Unfortunately I did not have an opportunity of seeing this urine, which was not examined for corpuscles. It seems probable that the rapid fall of worth which occurred soon after was connected with this event.

In the case of the child A. B., Chart VIII, the hæmoglobin estimation was also repeated with a fresh drop of blood on October 8th, when a rise of hæmoglobin was observed, and the two examinations gave identical percentages.

One or two of the charts suggest that possibly the salicylic treatment may have a share in the causation of the falls of worth (notably Chart VIII), but in other cases similar falls are seen which are quite independent of salicylic treatment, and I believe that this factor may be safely excluded.

Attempt to trace a Relation between the Changes in the Blood and the Appearance of Uro-hæmatoporphyrin in the Urine.

Uro-hæmatoporphyrin is an urinary pigment which was discovered and described by Dr. MacMunn, who has shown further that it is identical with one of the products of the action of reducing agents upon hæmatin, and is therefore almost certainly derived directly from hæmoglobin. I do not propose to enter here upon any lengthy discussion of the properties of uro-hæmatoporphyrin, nor of the morbid conditions under which it appears in the urine, for this would lead me far away from my present subject. I may, however, be allowed to call attention to certain points in connection with this substance. Uro-hæmatoporphyrin appears to be very closely related to hæmatoporphyrin, and it is permissible to doubt whether we are justified in

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regarding these bodies as absolutely distinct on the ground of the spectroscopic differences alone. In acid solutions uro-hæmatoporphyrin yields a spectrum containing two dark bands and an intermediate shaded band, which I have failed to distinguish, by measurement, from those which constitute the spectrum of acid hæmatoporphyrin in dilute solution, as when some of this substance is added to normal urine. Under such conditions the broad shading, which in concentrated solutions is continuous with the second band of acid hæmatoporphyrin, is reduced to a faint but distinct narrow band which corresponds in position with the intermediate shaded band of uro-hæmatoporphyrin.

Dr. MacMunn is convinced that a third band near the F line forms an essential part of the spectrum of acid uro-hæmatoporphyrin, but Le Nobel maintains that this line is due to impurities; and I may remark that it has seemed to me that the intensity of the band in question diminishes as greater purity is arrived at, and in some of my specimens it has been reduced to a mere shading, too indistinct for accurate measurement.

When a solution of uro-hæmatoporphyrin is rendered alkaline with ammonia it sometimes yields a five-banded spectrum, chiefly differing from that of hæmatoporphyrin in the presence of two bands instead of a single narrow band in the red.

I may mention that I have repeatedly prepared a substance giving spectra identical with those of the urinary pigment, in the same way as Dr. MacMunn has done, namely, by acting upon an alcoholic solution of hæmatin with zinc and sulphuric acid, and also by the reduction of ordinary hæmatoporphyrin. My own observations also support Dr. MacMunn's statement that uro-hæmatoporphyrin is usually present in considerable quantities in the urine of patients suffering from acute rheumatism, and that the pigment present in such cases frequently yields the characteristic five-banded alkaline spectrum. In some of my cases, however, a four-banded spectrum was

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obtained on the addition of ammonia. The chloroform extract from such a specimen was evaporated to dryness on a filter, and the residue dissolved in rectified spirit gave a five-banded spectrum, the readings of which agreed closely with those given by MacMunn for neutral uro-hæmatoporphyrin, and also with those of the spectrum of neutral hæmatoporphyrin, which also shows five bands. I am strongly inclined to think that in this case the pigment present was ordinary hæmatoporphyrin; and it is interesting to note that in this case alone did I succeed in obtaining an acid spectrum without any line at F.

Dr. MacMunn has also pointed out that in some cases the urinary pigment gives a four- instead of a five-band spectrum when rendered alkaline with ammonia.

The quantity of uro-hæmatoporphyrin present in the urine of rheumatic patients does not approach to the amount of hæmatoporphyrin or allied substances present in some cases which have been described by Stockvis,[1] Salkowski,[2] Ranking and Pardington,[3] and MacMunn,[4] in which the liquid had a deep red colour; indeed, the colour of the urine is not, as a rule, appreciably altered by the presence of the pigment, even after the addition of an acid. In some cases the acid spectrum may be seen after simple acidification, and in one instance I have seen some of the bands of neutral uro-hæmatoporphyrin without any preparation at all; in most cases, however, it is necessary to obtain a concentrated extract in order to observe the bands; and in some the quantity present is so small that it is not possible to obtain the alkaline spectrum. It must not be supposed that the presence of uro-hæmatoporphyrin in the urine is in any way pathognomonic of rheumatism; it is merely an unusually constant phenomenon in that disease, and the quantity present is often unusually large.

[1] 'Nederl. Tijdschrift voor Geneeskunde,' 1889, ii, p. 413.
[2] 'Lancet,' 1890, vol. ii, p. 607.
[3] 'Zeitschrift für physiol. Chemie,' Bd. xv, p. 286, 1891.
[4] 'Journal of Physiology,' 1890; 'Proc. Physiol. Soc.,' p. xiii.

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The method employed for the detection of uro-hæmatoporphyrin in the cases referred to in this paper was that recommended by Dr. MacMunn.

Neutral and tribasic acetate of lead were added to the specimens of urine, and the precipitate formed was filtered off from the decolourised liquid. The precipitate was then treated with rectified spirit acidulated with sulphuric acid, and the extract so obtained was examined with the spectroscope.

It is unfortunately only possible to form a very rough estimate of the relative amounts of the pigment present in different specimens; but when equal quantities of urine are examined, and nearly equal quantities of acidulated alcoholic extract are prepared, it is possible to obtain some idea of the quantity present from the relative intensity of the absorption bands in different specimens.

It seemed probable that, since uro-hæmatoporphyrin is a reduction product of hæmatin, it might be possible to trace some relation between its appearance in the urine and the destruction of red corpuscles or of hæmoglobin. I accordingly made frequent examinations of the urine in seven cases, in which I was at the same time observing the changes in the blood, and I am obliged to acknowledge that I have failed to detect any obvious connection between the blood-changes and the excretion of this pigment.

In the first case examined from this point of view (Chart VII) it seemed as if the appearance of uro-hæmatoporphyrin in the urine was associated with falling worth, but the later observations lent no support to this view, for the pigment was present in some cases whilst the worth was rising, and the repair of the red corpuscles was going on rapidly.

Chart IV exhibits only very trifling variations of worth, but nevertheless uro-hæmatoporphyrin was found on no less than fourteen out of the seventeen occasions on which the urine was examined, sometimes in mere traces, at other times in considerable quantity.

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Again, in the case represented by Chart VIII uro-hæmatoporphyrin was found repeatedly, in varying amounts, both during the period of deterioration of the blood and during that of repair.

Chart VI is less conclusive. In this case the excretion of uro-hæmatoporphyrin ceased when convalescence was fairly established, and only at the first two observations was any considerable quantity found.

In some cases in which the blood-changes are well marked no uro-hæmatoporphyrin is found in the urine.

Chart III illustrates this; here the variations in the number of red corpuscles are most conspicuous, but the urine was examined on five occasions, always with negative results.

Perhaps the absence of any relation between the two sets of phenomena was most strikingly demonstrated in the case of a patient whose chart I have not reproduced. He was a man, æt. 34, who on his admission to the hospital was passing urine which contained a large amount of uro-hæmatoporphyrin, or most probably ordinary hæmatoporphyrin. During his stay of twenty-one days nine examinations of the urine were made, and the pigment was found to be present on each occasion. He was discharged on August 31st, the corpuscles having increased 17 per cent. since his admission, and the hæmoglobin 6 per cent., whereas the worth of the individual corpuscle had fallen ·06. On September 23rd he attended as an out-patient with some return of pain in the joints, and his urine was found still to contain hæmatoporphyrin. On September 26th the pigment was again present in large quantities, as also on October 10th, when the patient was readmitted with a moderately acute rheumatic attack. Hence it may be inferred that the patient was excreting hæmatoporphyrin during at least a considerable part of the interval between his discharge and readmission, but yet on October 12th the red corpuscles numbered 4,793,750 as against 4,918,750 at the time of his discharge; the percentage of hæmoglobin

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had risen from 67 to 71, and the worth of the individual corpuscle from ·68 to ·74.

It may be that there is a relation between the changes in the blood and urine which the methods employed are not sufficiently exact to detect, but I do not think the observed phenomena can be in any way explained by supposing that just as drugs are excreted in the urine for some time after their administration is stopped, so uro-hæmatoporphyrin may appear in the urine after the process which leads to its formation has come to an end. In Case 8, for instance, the pigment was still found in the urine twelve days after the last evidence of excessive blood destruction was obtained; for the fall of worth between October 8th and 12th would seem to have been due rather to a deficiency of hæmoglobin in the freshly formed corpuscles than to any active destructive process.

I think, indeed, that the above-quoted cases afford sufficient evidence that the presence of uro-hæmatoporphyrin in the urine does not imply any excessive destruction of red corpuscles or of hæmoglobin, and that, unless it be derived from the histohæmatin of muscle, its excretion must be attributed to a perversion of the ordinary processes for the disposal of effete blood-pigment rather than to a destruction of larger quantities of hæmoglobin than the ordinary channels of elimination are capable of dealing with. At the same time it is a fact that the period of rheumatic fever, in which uro-hæmatoporphyrin is most apt to be present in the urine, is one during which the processes of blood destruction are very active at times.

Indications for the Treatment of the Anæmia of Rheumatism.

It will be seen from the observations which are embodied in the paper that the anæmia of rheumatism is of two kinds: 1st, an acute oligocythæmia developing during the acute stage of the disease, and rapidly recovered

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from as soon as convalescence sets in; and 2ndly, a pseudo-chlorotic condition which is developed as a sequela of the attack in a few cases, and which, unless it is appropriately treated, may last for a long period, the fall of worth continuing in spite of the amelioration of the patient's condition in other respects.

The acute oligocythæmia calls for no special treatment, but the fall of the number of corpuscles in some cases, and of the hæmoglobin percentage in others, which appears to be dependent upon the patient's first getting up, suggest the desirability of keeping the patient in bed for some time after the subsidence of the acute symptoms, even when it is not rendered especially desirable by an affection of the endocardium. Iron appears to be often extremely useful in combating the pseudo-chlorotic condition. Its effect was well shown in the little boy, aged eight, who was left conspicuously anæmic after an attack of articular rheumatism associated with chorea and extremely numerous subcutaneous nodules, and who had a loud præsystolic mitral murmur. On June 20th this boy's corpuscles numbered 5,012,500, but his hæmoglobin percentage was only 60, the worth of the individual corpuscle being ·59. He was given two drachms of steel wine three times a day, and on July 4th the red corpuscles had increased to 5,634,670, the hæmoglobin to 75 per cent., and the worth to ·66. On July 17th, another fortnight later, the corpuscles had again fallen to 4,968,750, but the hæmoglobin remained at 75 per cent., and the worth had further risen to ·75, making an increase of ·16 in four weeks.

It should be mentioned that even at the last-mentioned date the nodules had not all disappeared, and fresh ones occasionally developed for some time afterwards.

Again, it would appear that, in the case represented in Chart V, the substitution of fifteen grains of the ammonio-citrate of iron daily for sodium salicylate checked the fall of worth which was till then progressing rapidly; but, on the other hand, Chart VII shows no

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effect from the administration of similar doses of the same compound, at least during the period over which the observations extend.

Note.—Since the above was written further study of the properties of the urinary pigment referred to has convinced me that it is actually hæmatoporphyrin, and that its spectrum in acid solution free from urobilin is identical with that of hæmatoporphyrin prepared from blood, showing no band before the F line.

Part II.—*Variations in the Number of White Corpuscles. The Character of the Fibrin Network in Simple Slide Preparations of the Blood.*

Received March 3rd—Read May 24th, 1892.

Variations in the Number of White Corpuscles.

In the first part of this paper I have described in detail the changes which I observed in the number and worth of the red corpuscles in the course of rheumatic attacks; and I now propose to supplement that description by an account of the variations in the number of leucocytes which occur simultaneously with those changes.

M. Hayem[1] states that the white corpuscles are always increased in number during acute attacks, but Drs. Maragliano and Castellino[2] maintain that there is no real leucocytosis in these cases, and attribute the apparent increase of the white to the relative diminution of the red corpuscles.

Mr. Gostling,[3] in his important paper on the increase

[1] 'Du Sang' (1889), p. 916.

[2] 'Gazzetta degli Ospitali,' 22 Marzo, 1891.

[3] 'Medico-Chir. Trans.,' lxix, 1886, p. 206.

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of white corpuscles in the blood in inflammation, communicated to this Society in 1886, quotes only the numbers counted in a single case of rheumatic fever, and contrasts the scanty fluctuations there observed with the far more conspicuous variations which are associated with suppurative diseases. Halla,[1] v. Limbeck,[2] Reinert,[3] and Carl Sadler[4] have also recorded cases in which some increase of the number of white corpuscles was observed.

My own observations tend, one and all, to confirm the accuracy of M. Hayem's statement upon this point. In every case of acute rheumatism in which I have examined the blood there has been evidence of a distinct increase of white corpuscles during the febrile stage, but at the same time the leucocytosis has been very moderate in degree.

For the counting of the white, as well as of the red corpuscles, a Thoma-Zeiss hæmocytometer was employed, with the special mixing pipette which is made for this purpose, giving a dilution of only ten or twenty instead of one or two hundred times.

The blood was diluted with a ·03 per cent. solution of acetic acid, a liquid which is very convenient for the purpose, as it renders the leucocytes more conspicuous, and at the same time destroys the red corpuscles.

A microscopic field of known area was taken as the unit, and fifty such fields were counted at each observation. The area of the field taken was such that the cubic capacity of the portion of the hæmocytometer-cell included in fifty fields was equal to that over 490·8 squares of Gowers' hæmocytometer.

It appears to me to be the better plan to state the number of leucocytes per cubic millimetre of blood, rather than to give merely the relation between the numbers of white and red corpuscles as is usually done. The relative

[1] 'Zeitschr. f. Heilkunde,' 1883, Bd. iv, p. 348.

[2] Ibid., 1890, Bd. x, p. 422.

[3] 'Die Zählung der Blutkörperchen,' &c., 1891.

[4] 'Fortschritte der Med.,' Bd. x, 1892, 4, Supplement Heft.

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numbers have, of course, the advantage of being independent of changes in the total mass of blood, but they give but a very imperfect notion of the fluctuations of the white corpuscles, which may be almost completely masked if the numbers happen to be increasing or diminishing simultaneously with those of the red corpuscles.

The average number of white corpuscles contained in healthy blood has been very variously estimated; but my results agree most closely with those of more recent observers, who give, for the most part, a considerably lower figure than was formerly adopted.

M. Hayem considers that 6000 white corpuscles per cubic millimetre is about the normal; and Dr. Habershon, after making 80 examinations of the blood of forty healthy individuals between the ages of twenty and fifty, obtained an average of 6600 white corpuscles per cubic millimetre, or one white to 670 red corpuscles.[1]

Dr. Habershon also found that when the blood was examined at a short interval after a meal the average was somewhat higher, viz. 8620 per cubic millimetre.

In order to exclude the effects of food as far as possible, my examinations were made at nearly the same hour on different days, usually between 3 and 4 o'clock in the afternoon, the chief meal having been taken at 1 p.m.

It is upon the fluctuations observed, rather than upon the actual number of white corpuscles present at any given period, that I rely for the proof of the statement that there is a true leucocytosis in acute rheumatic cases, for there are few readings of which I have records which would not be regarded by some as but little exceeding the numbers compatible with health.

For the sake of convenience I have taken 7000 per cubic millimetre as the normal number, and have called this 100 per cent., and have thus been able to represent the variations on the charts by making this number correspond with the zero line.

[1] "On the Variations in the Number of White Blood-corpuscles in Diabetic Patients," 'St. Barth. Hosp. Reports,' vol. xxvi, 1890, p. 153.

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The conclusions here presented are drawn from a series of eight cases in which the special white corpuscle pipette was employed; but I may add that in a number of other cases, in which the white corpuscles were counted with the red, results were obtained entirely in accordance with those arrived at by the more exact method.

M. Hayem mentions 25,000 per cubic millimetre as the number to which the leucocytes may attain in very acute cases, but none of the cases which I examined in this connection could be described as very acute, and the highest observed number was 19,882, or 284 per cent., the proportion of white to red corpuscles being as 1 to 209.

In the first part of this paper it was pointed out that the diminution of the number of red corpuscles had but little relation to the amount of the febrile disturbance, having a far more obvious association with the occurrence of local lesions. The increase of leucocytes, on the other hand, seems to have an intimate relation to the height of the fever, whereas local lesions attended with little rise of temperature may be accompanied by an increase of white corpuscles so slight as hardly to exceed the limits of health.

These points are well illustrated by the case of a patient whose chart is numbered III. In that instance a copious eruption of erythema marginatum, which appeared during convalescence from a slight attack of articular rheumatism, was attended by a loss of no less than 1,000,000 red corpuscles per cubic millimetre, whereas the maximum number of leucocytes counted, at a time when the red corpuscles were at their lowest, was only 8271 per cubic millimetre, or 118 per cent.

The increase of white corpuscles is a very early event in the rheumatic attack, so early, indeed, that it has usually reached its limit before the first observation is made; and in hospital patients it is only possible to observe the fall which occurs as soon as convalescence commences.

When, however, a relapse or recrudescence occurs

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under observation the leucocytosis may be satisfactorily studied; this is very well illustrated by Chart IX.

The patient in question was a young woman, æt. 21, who had been in the West London Hospital a year previously, suffering from acute articular rheumatism and valvular disease, and who then exhibited numerous subcutaneous rheumatic nodules.

She was admitted a second time on November 15th, 1891, under the care of my colleague Dr. Herringham, to whom I am greatly indebted for the opportunity of studying this and other cases.

The primary attack quickly subsided under treatment, and the white corpuscles, which on November 16th numbered 11,714 per cubic millimetre, or 167·5 per cent., had fallen by November 19th to 9160, or 133·3 per cent., whilst there had been a simultaneous recovery of red corpuscles.

On the morning of November 23rd the temperature, which had been normal, was found to have risen again to 100°, and in the afternoon of that day there was found to be a suspicious increase of white corpuscles, although the number of red corpuscles was also increased.

This rise of temperature marked the commencement of one of those remarkable relapses in which local manifestations are almost entirely wanting, although the febrile disturbance is considerable. There was, indeed, a slight return of articular pain, but this was unattended by any swelling, and there was no evidence of any rapid increase of the cardiac mischief, nor of the development of endocarditis; nevertheless it was, of course, impossible to exclude the possibility of fresh endocarditis.

On November 26th the white corpuscles had undergone a further increase to 15,217 per c.m., or 217 per cent., and there was a moderate loss of red corpuscles.

The temperature remained high, with occasional intermissions, until December 5th, but on November 29th the white corpuscles had already fallen to 11,041, or 157·7 per cent., and on December 3rd a further diminution to 10,369,

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or 148·1 per cent., was recorded. On the 7th the number was 8780, or 125·4 per cent.; but on December 12th, although the temperature continued to be normal and subnormal, a fresh increase of white corpuscles to 9595 was found to have taken place, and this increase was the earliest warning of a second relapse, during which the blood was not further examined.

The decrease of the white corpuscles during the decline of the attack may be as rapid as their increase at its onset, as is shown by the following examples:

Date.		Number of white corpuscles per cm.		Percentage.
Case 1.—Oct. 29th	...	17,194	...	245·6
Nov. 2nd	...	7,700	...	110·0
Case 2.—Jan. 23rd	...	19,882	...	284·0
„ 27th	...	17,950	...	256·4
„ 30th	...	9,024	...	128·9

On the other hand, there are cases in which the fall is very gradual, and the number of white corpuscles may remain high for some time after the febrile disturbance, and all obvious local manifestations of the disease have come to an end. The following case illustrates this:

Date.		Maximum temp. of day.		Number of white corpuscles per cm.		Percentage.
Oct. 8th	...	100·8° F.	...	13,300	...	190·0
„ 12th	...	99°	...	12,691	...	181·3
„ 17th	...	98·4°	...	12,831	...	183·3

The Blood Platelets.

Since the blood platelets remain visible for a considerable time in the solution of sodium sulphate, they may be observed and counted on the hæmocytometer field. In some cases in which I have attempted to count them there has been a well-marked increase in their numbers during the period of convalescence, and in one instance the increase was very remarkable, the number of platelets reaching the very high total of 1,415,000 per cubic millimetre, or nearly one platelet to every three red corpuscles.

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Halla[1] also observed a great increase of platelets after defervescence in some of his cases.

Form of the Red Corpuscle.

I have not observed any noteworthy change in the form of the red corpuscles, and in the cases in which they were measured with a micrometer scale no marked deviation from the normal size was noticed. In a few cases in which the degree of anæmia was conspicuous, some distortion of shape was apparent in some of the corpuscles.

Characters of the Fibrin Network.

When a drop of blood from a rheumatic patient is placed upon a slide and simply enclosed by a cover-glass, the corpuscles soon become massed together, and lose their natural shape, the masses of corpuscles being separated from each other by broad lakelets of clear serum, in which lie numerous leucocytes. In a short time filaments of fibrin, much coarser than the delicate fibrillæ which form in healthy blood, begin to traverse the clear spaces in all directions, and in this way a coarse fibrin network is gradually formed, which has a radiating structure, the radiant points being formed by groups of leucocytes or collections of blood platelets.

M. Hayem[2] considers that these appearances of the blood have a very important diagnostic value, since he has found that the fibrin network is more conspicuous in acute rheumatism than in any other disease except croupous pneumonia.

In order to test the diagnostic value of this indication, I have examined in the manner described the blood of a number of patients suffering from a variety of inflammatory disorders, and have been led to the opinion that the test has only a very limited application. Undoubtedly

[1] 'Zeitschr. f. Heilkunde,' 1883, Bd. iv, p. 349.

[2] 'Bull. et Mém. Soc. Méd. des Hôpitaux,' 1886, p. 18.

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the above description agrees with what is observed in the great majority of cases of rheumatic fever, and in the patient whose chart is numbered IX the character of the fibrin network varied, as will be seen, with the intensity of the rheumatic manifestations, and especially with the degree of febrile disturbance, the network becoming much more delicate when the temperature fell, and much coarser again when the relapse set in. Yet it occasionally happens that the blood exhibits a different appearance, even in typical rheumatic cases, as is shown by the following example.

A young woman was admitted to the hospital suffering from acute arthritis of many joints, and having a loud pericardial friction-sound. The blood was examined on the day following her admission, and was found to present a quite unusual appearance. The corpuscles very rapidly became crenated, and did not show the slightest tendency to form rouleaux or to aggregate into masses; indeed, no one corpuscle adhered to any other. There were no masses of corpuscles and no lakelets of serum, nor could any fibrin network be seen with a quarter-inch power, even after an interval of half an hour. The morning temperature on the day in question was 99·8°, the evening reading 102·4°.

These unusual appearances suggested a doubt as to the accuracy of the diagnosis of rheumatic fever, but the subsequent course of the case, its behaviour under salicylic treatment, and the development of an endocardial murmur dispelled all question as to its nature. Four days later the blood of this patient presented an appearance more closely resembling that usually met with in rheumatic cases, but the temperature was falling, and the fibrin network, although conspicuous, was by no means coarse.

I do not doubt that the appearance of the blood may be of considerable value in cases in which febrile disturbance precedes any of the more characteristic rheumatic manifestations, by giving a useful hint as to the probable nature of the disease, and it is in such cases that

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M. Hayem has found it of service; but in afebrile cases, when the question arises whether or no local lesions which are sometimes rheumatic are in the particular instance manifestations of rheumatism, it is of no service, since the coarse network is wanting, even in attacks the rheumatic origin of which is almost beyond question, as long as the temperature exhibits no considerable elevation.

Again, in cases of acute gout the blood may exhibit appearances which only differ very slightly, if at all, from those met with in acute rheumatism; and hence the test is of no service in distinguishing between these two forms of articular affection, which sometimes resemble each other so closely.

(For reports of the discussions on these papers, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, pp. 70 and 132.)

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CHART I.—Emily P—, æt. 18. Rheumatic fever; arthritis; pericarditis; endocarditis; erythema nodosum.

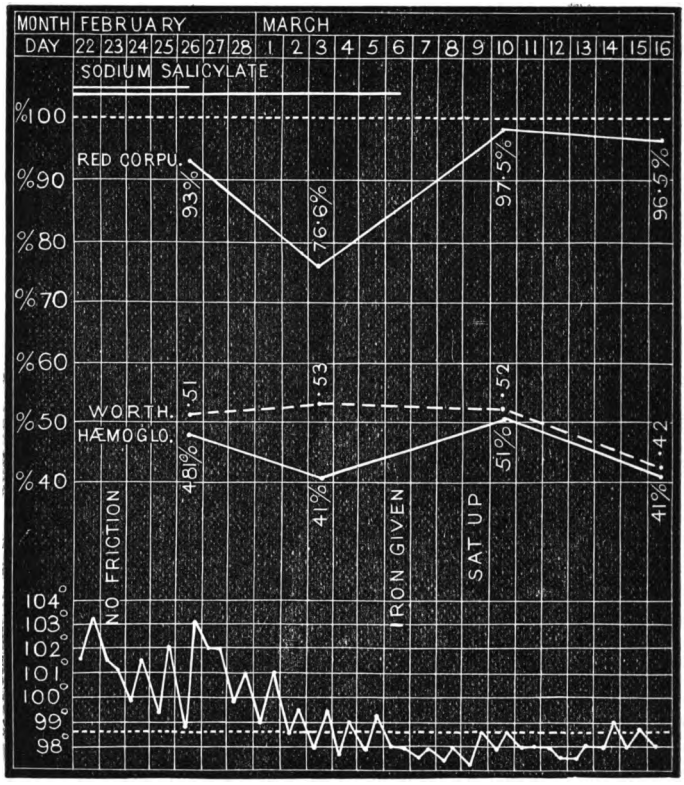

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CHART II.—H. G—, male, æt. 16. Rheumatic fever; pericarditis; pneumonia; pleurisy.

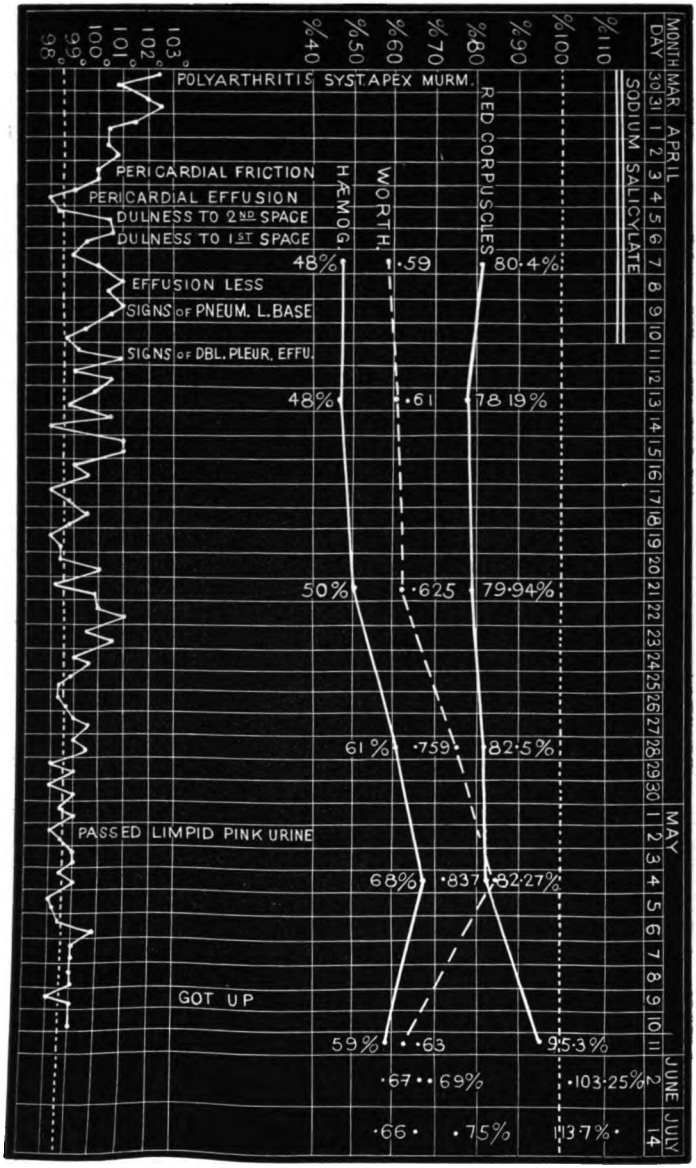

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CHART III.—W. B—, male, æt. 18. Subacute rheumatism; pericarditis; erythema marginatum; nodules.

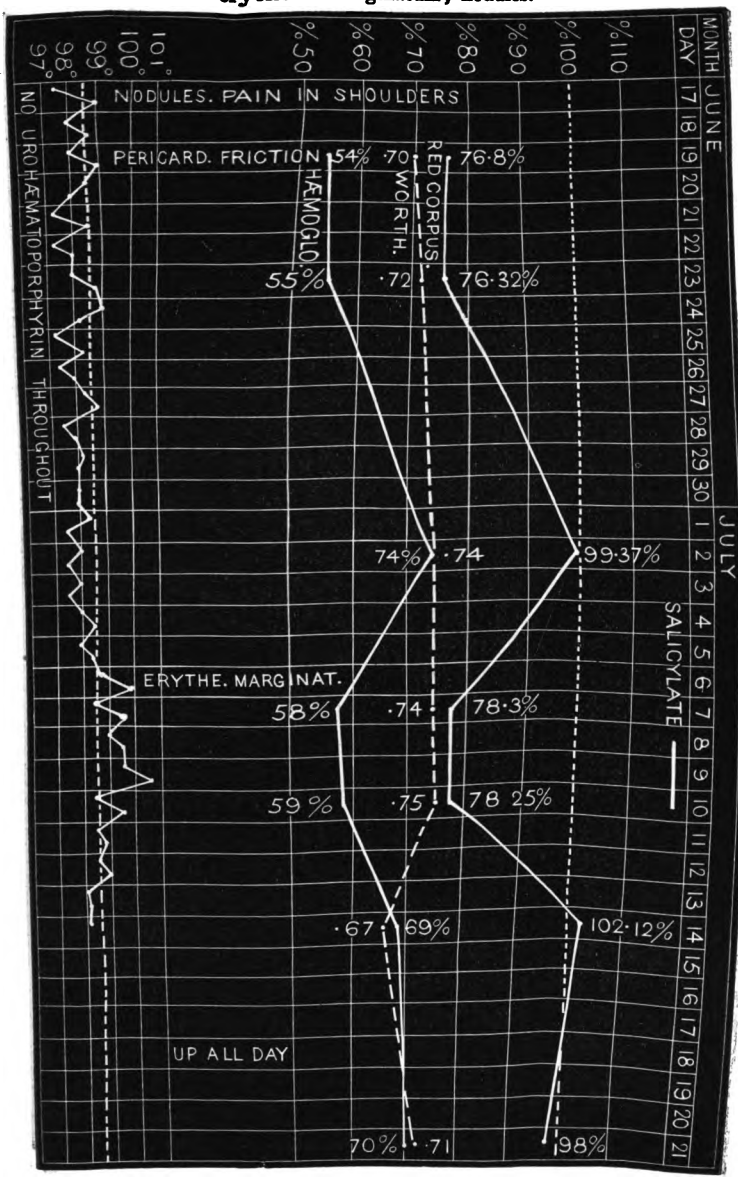

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CHART IV.—W. F—, male, æt. 17. Subacute rheumatism; pleurisy.

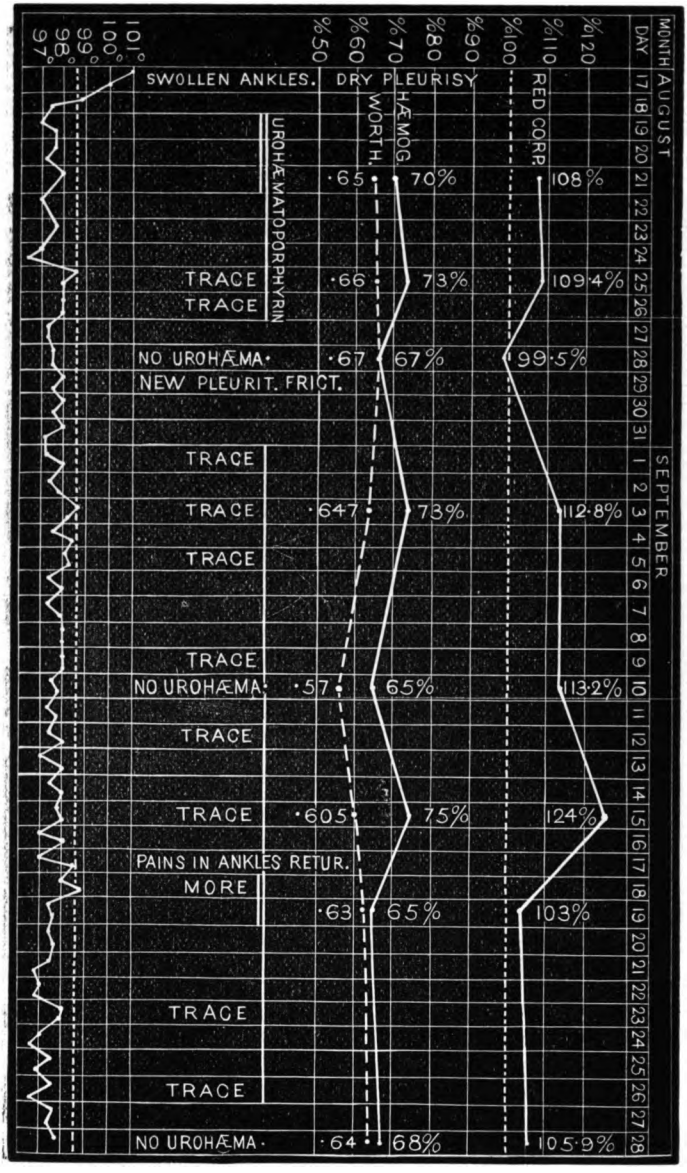

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CHART V.—F. H—, male, æt. 18. Rheumatic fever; pericarditis.

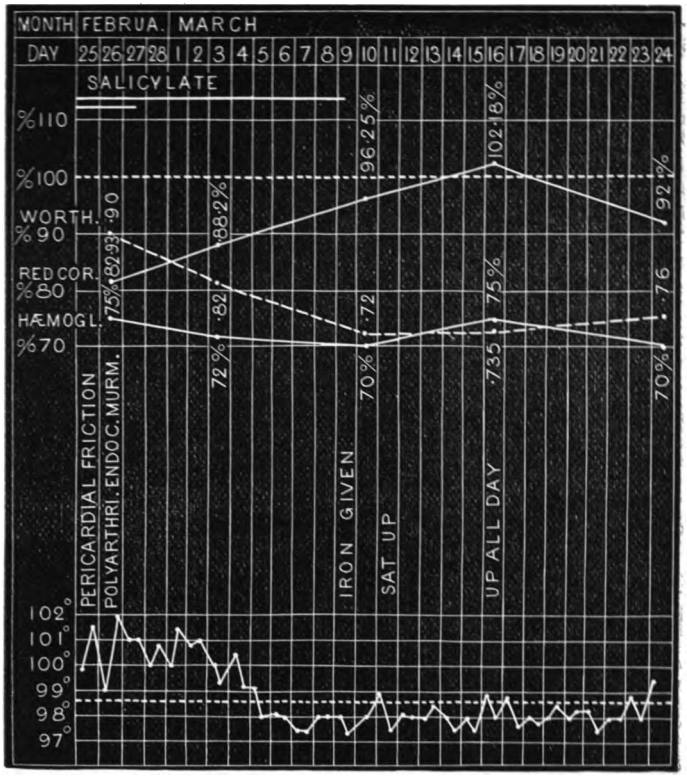

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CHART VI.—C. T—, male, æt. 9. Subacute rheumatism; arthritis; endocarditis.

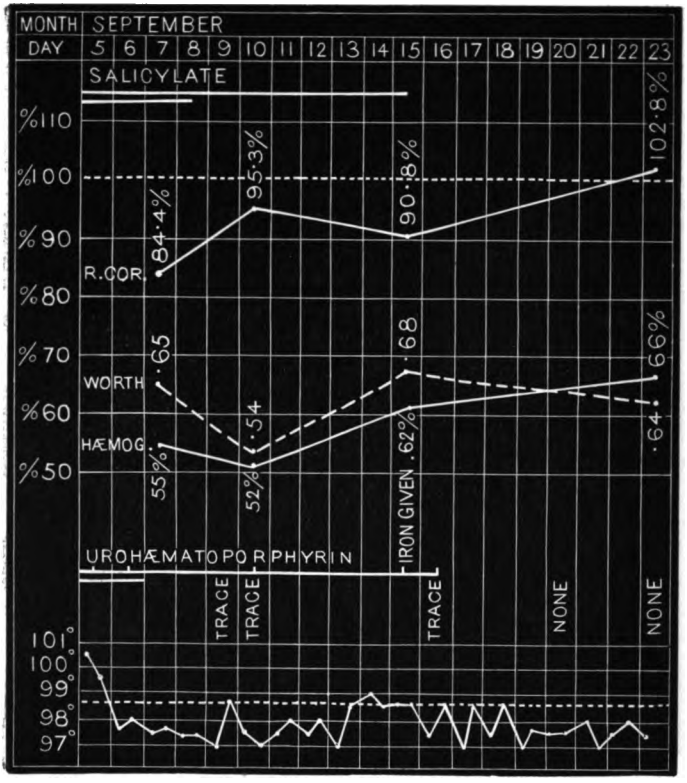

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CHART VII.—H. R—, male. Rheumatic fever; arthritis; endocarditis.

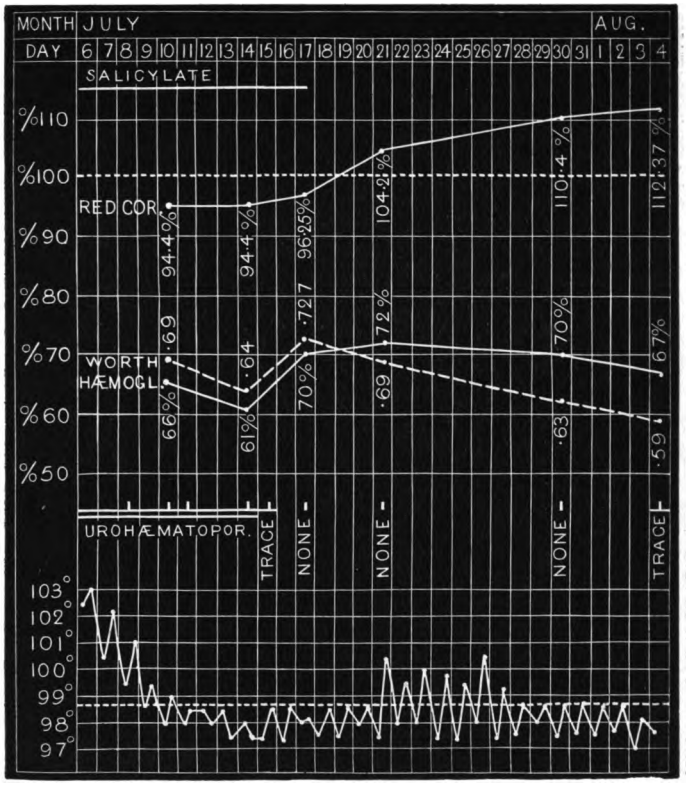

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CHART VIII.—A. B—, female, æt. 10. Rheumatic fever; endocarditis; bronchitis.

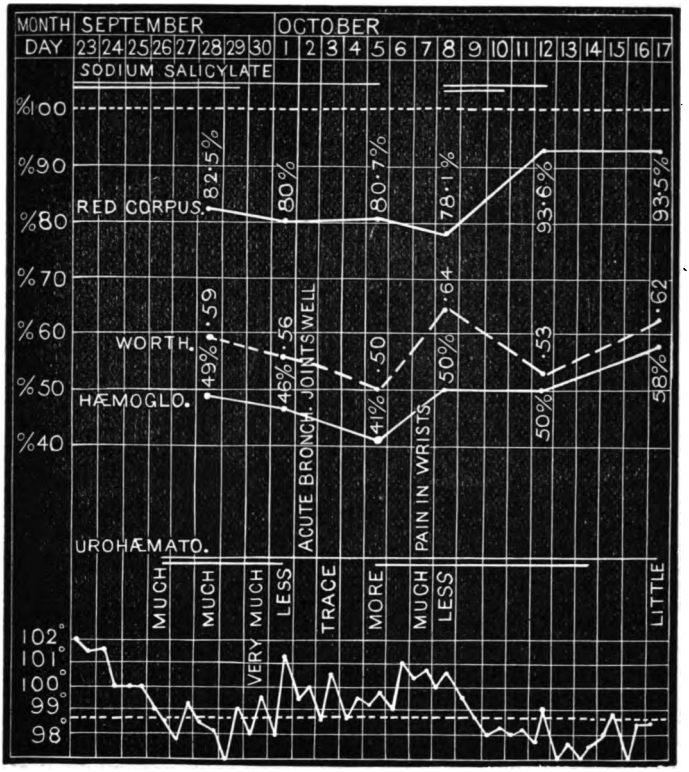

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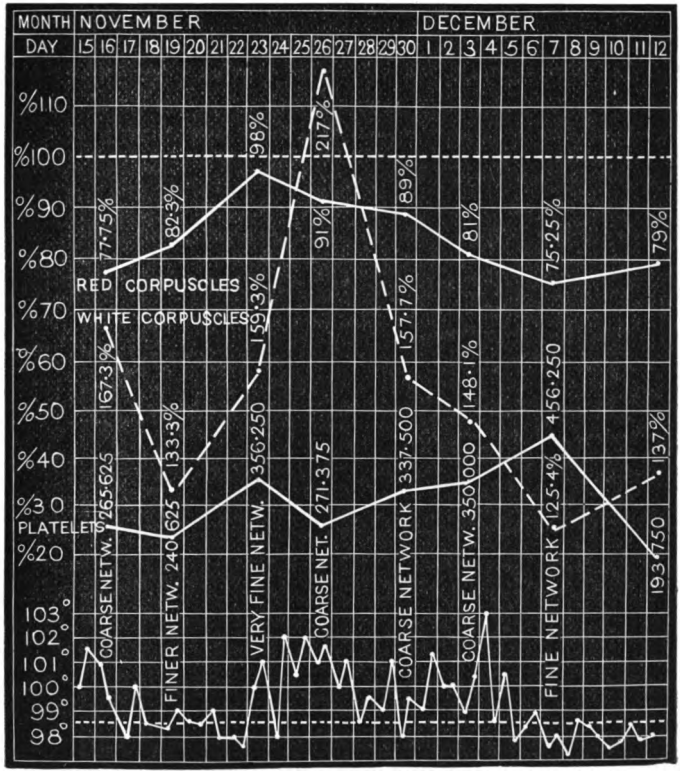
CHART IX.—F. C—, female, æt. 21. Rheumatic fever; relapse.



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"ANTISEPTIC" PREPARATIONS OF CATGUT AND SILK;

THEIR RELATION TO WOUND INFECTION.[1]

BY

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(PRESENTED BY MR. BOWLBY.)

Received November 24th, 1891—Read February 23rd, 1892.

IN the Bradshaw Lecture, delivered by Sir Spencer Wells in December, 1890, allusion[2] was made to certain experiments, which were then being conducted by me at the Research Laboratories of the conjoint Colleges of Physicians and Surgeons. These experiments are now sufficiently completed to enable me to bring forward certain definite facts, which may throw some light upon the occurrence of suppurations in wounds treated apparently in strict accordance with antiseptic principles, and upon the aseptic course of others treated apparently without these precautions.

It is unnecessary here to do more than allude to the

[1] This paper is a short account of experiments conducted at the research laboratories of the conjoint colleges during the winter of 1890-91.

[2] 'British Medical Journal,' Dec. 20th, 1890, p. 1418.

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fact that suppurations, often of a severe character, arising from the presence of ligatures and buried sutures have been frequently noted. It is only, however, within recent years that special attention has been directed to the actual presence of septic organisms in the material so employed. Zweifel[1] was amongst the first to draw attention to this point, and Sir Joseph Lister[2] has himself referred to an interesting instance occurring in his own practice in connection with the use of the old hempen ligature. It is Kocher, however, who has specially emphasised the importance of employing absolutely aseptic ligature and suture material, and of clinically observing the behaviour of material preserved in apparently trustworthy antiseptic solutions. The history of his practice, so far as it bears upon this, is most instructive. Shortly after Zweifel drew attention to the point, he traced septic complication in one of his cases to the carbolised catgut employed in the operation, and on opening several new bottles of carbolised gut he found that they had a putrid smell, similar to the smell of the pus in the case referred to. This may have been caused by originally contaminated gut, but, as Kocher remarks, the fact remains that its preservation in 20 per cent. solution of carbolic acid in oil, and before use in 5 per cent. aqueous solution was insufficient to render it aseptic.[3] He consequently discarded catgut and used silk steralised according to Czerny's method.[4] In 1881, in consequence of his well-known experiments with juniper oil,[5] he commenced using catgut and silk treated with juniper oil and preserved in alcohol. In 1888,[6] however, he tells us that he has again discarded catgut, and finally adopted silk for all operations, preserving it in 1 in 1000 solution of mercuric

[1] 'Centralb. f. Gynäk.,' 1879, iii, p. 295, "Catgut als Träger der Infection."

[2] "Inaugural Address," 'Trans. Clin. Soc.,' vol. xiv.

[3] 'Centralb. f. Chir.,' 1881, p. 353.

[4] 'Beiträge zur Operativ. Chir. Enke.,' 1878, p. 11.

[5] "Zubereitung von antiseptischen Katgut," loc. cit.

[6] "Eine einfache Methode zur Erzielung sicherer Asepsis," 'Correspondenzb. f. Schweizer Aerzte,' 1888, No. 1, p. 3.

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chloride. This change of practice was due to the fact that out of a series of 31 major operations, performed during a period of seven weeks, he had 22 cases of suppurative wound infection distinctly traceable to the catgut employed, whereas in a series of 62 subsequent operations, in which silk alone was used, there was not a single bad result.

Reference must also be made to an exhaustive and elaborate article on "Catgut Infection," by Dr. Conrad Brunner, of Zurich, published last year;[1] an article which is the most important that has yet appeared on the subject, but which I was unfortunately unable to obtain for perusal until after completing my experiments. It is noteworthy, however, that the results of similar experiments, conducted by him, are to a great extent confirmed by mine; a fact to which reference will be made later on.

My experiments were commenced in October, 1890, with a view to testing by bacteriological methods the so-called antiseptic preparations of manufacturers, and for the purpose of discovering, if possible, a simple and efficient method of ensuring absolute purity of ligature and suture materials at the time of their use. This latter point is of vast importance to surgeons who may have to undertake operations without the means of sufficiently steralising their material at the time, and has a special bearing on the surgical equipment of armies in the field; while the results of my experiments on manufacturers' preparations may be of interest and value to those who use such preparations under the impression that they are actually what they are said to be, and, therefore, in no need of further sterilisation.

The experiments were conducted in three series:—(1) Experiments testing the purity of dry material supplied by surgical instrument makers; (2) experiments testing the purity of material preserved in "antiseptic" solutions; (3) experiments with specially contaminated silk,

[1] 'Beiträge zur klin. Chir. von Brunsek.,' 1890.

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to test the antiseptic value of the solutions in which the material is usually preserved.

Control experiments were made; and, with a view to eliminating sources of error, the breath and hands of the operator, as well as the air and dust of the room, were examined as to the presence of micro-organisms at various periods. An inoculating box was used, whenever this was possible, and the instruments employed (scissors and forceps) were sterilised by heat in the usual manner. The material used was tested by placing pieces about half an inch in length in sterilised meat infusion, and incubating for at least three weeks, often much longer, at a temperature of 37° C. In all doubtful cases, and in many others, stroke and puncture cultivations were made from the meat infusion on nutrient gelatine, and any resulting growths examined under the microscope. As regards comparitive results, these latter observations have no bearing on the purity or otherwise of the material, and, therefore, for the sake of brevity, no detailed statement of them will be made here. Cultivations could only be obtained with difficulty from minute particles of dust in the room, and negative results were obtained after coughing and breathing into the tubes of nutrient material. Exposure of the tubes to the air, with the cotton-wool plugs removed, for one or two minutes also gave negative results; so that it may be assumed that the condition of the room was highly satisfactory for the carrying out of these tests, and that wherever the nutrient material showed putrefactive changes, these changes were due to putrefactive organisms originally adherent to, and introduced by, the silk or catgut.

First Series.—*Dry Preparations.*

The materials tested under this head were sulphurochromic gut, surgeon's silk, untreated by any antiseptic method, and "carbolised silk" stated to have been prepared under the instructions of Sir Joseph Lister. Two

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experiments were also made with plated wire; and a comparative test was made with "button-hole" silk, similar to surgeon's silk, but taken from the table of a constantly occupied sitting-room. The results of these experiments are shown in the following table.

TABLE I.—*Experiments with Dry Material.*

No. of experiment.	Nature of material.	Where obtained.	Date of experiment.	Result.
			1890	
1	Sulphurochromic gut	Army Med. Stores, Woolwich (from bearer's dressing case of field surgical haversac)	Oct. 24	Haziness of broth; no definite putrefaction. Puncture cultivation Nov. 24, 1890, showed no growth on April 4, 1891. Stroke cultivation Nov. 24, 1890, showed single colony on Feb. 6, 1891.
2	,,	,,	Nov. 5	Haziness; no definite putrefaction. Stroke and puncture cultivations on Nov. 24, 1890, showed no growth on April 4, 1891.
3	,,	Field Med. Companion (new) at Auxiliary Hosp., Woolwich	Nov. 28	Wrinkled pedicle formed in 2 days. Fluid remained clear.
4	,,	,,	,,	Permanently clear.[1]
5	,,	,,	Dec. 1	Permanently clear.
6	,,	,,	,,	Slight haziness. No definite putrefaction.
7	Surgeon's silk (twist), untreated	,,	Oct. 24	Permanently clear.
8	,,	,,	May 11	Permanently clear.
9	,,	Surgeon's pocket case, in daily use	Oct. 24	Hazy Oct. 27; turbid Oct. 31. Rapid liquefying growths in gelatine cultivations.
10	Surgeon's silk (plaited), untreated	Reel, in case of instruments at Med. Dept., Royal Arsenal	Nov. 28	Permanently clear.
11	,,	,,	Nov. 28	Permanently clear.
12	"Carbolised silk" (medium size)	Surgical instrument maker	Oct. 24	Turbid Oct. 27; pedicle Oct. 29. Rapid liquefying growths in gelatine cultivations.

[1] Tubes noted as remaining "permanently clear" were under observation for periods varying from one to three months.

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TABLE I (continued).

No. of experiment.	Nature of material.	Where obtained.	Date of experiment.	Result.
13	"Carbolised silk" (medium size)	Surgical instrument maker	Oct. 29	Turbid Oct. 31, 1890. Rapid liquefying growths in gelatine cultivations.
14	"	"	"	Turbid Oct. 31, 1890. Rapid liquefying growths in gelatine cultivations.
15	"	Manufacturer of antiseptic dressings	1891 Jan. 30	Turbid Feb. 4, 1891.
16	"	"	"	Turbid Feb. 4, 1891.
17	Same material, but finest size	"	"	Permanently clear.
18	"	"	"	Permanently clear.
19	"Button-hole" silk, similar to surgeon's twist silk (untreated)	Skein of silk in occupied sitting-room	1890 Nov. 28	Wrinkled pedicle Nov. 29, 1890. Fluid remained clear.
20	"	"	"	Clear till Dec. 12, 1890; then slight turbidity.
21	"	"	Dec. 1	Wrinkled pedicle Dec. 3, 1890. Turbid Dec. 8, 1890.
22	"	"	"	Turbid Dec. 3, 1890.
23	Plated wire	Case of instruments in Medical Dept. of Royal Arsenal	Oct. 24	Permanently clear.
24	"	"	Nov. 5	Permanently clear.

Remarks.—The sulphuro-chromic gut and the surgeon's silk of experiments 7 to 11, as well as the plated wire, had been kept in instrument cases on reels or cards, and had been placed and kept in ordinary paper envelopes after removal from the cases. The "carbolised silk" was rolled on a glass rod attached to a cork stopper and kept in glass bottles. Of all the material shown in the table, the "button-hole" silk had been the most exposed

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to contamination by dirt or dust, and the "carbolised silk" the least.

It is a remarkable fact that of the material shown in Table I, the only material subjected to any "antiseptic" method of preparation by manufacturers was undoubtedly the most septic, if we except, that is to say, the "button-hole" silk, which had not been intended for surgical use.

The silk in the surgeon's pocket case was expected to prove septic, because it had been exposed to contamination in many places at home and abroad almost daily during a period of eight years, and consequently the only other experiments made with it were with a view of sterilising it. This was readily effected by boiling for one hour, and also by placing it for one hour in 1 in 4000 solution of mercuric chloride. I may mention here that the same processes were applied to portions of other material, which proved septic in these experiments, and in all the cases the material was rendered aseptic by them.

The results of the experiments with "carbolised silk" (which is, I believe, silk coated with a mixture of bees-wax and carbolic acid, prepared, as stated on the bottle, according to the instructions of Sir Joseph Lister) show that this method of treatment offers not only no guarantee that the silk is aseptic, but points rather to the probability that septic organisms become permanently adherent to the silk during the process. Experiments are detailed below (*vide* Tables IIIA and IIIB), which show how liable manufacturers are to miss the point in preparing aseptic material. At the same time, the coating of ligatures with viscid substances, however "antiseptic" they may seem to be, is objectionable, for the manifest reason that micro-organisms are more liable to adhere to such substances than to untreated material. The proposal by Dr. J. Craig[1] to coat silk with shoemaker's wax because it is "highly antiseptic" is a case in point.

The results of experiments 7, 8, and 10, 11, revealed

[1] 'New York Med. Journ.,' vol. xlii, 1885, p. 611.

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what was to me a new fact, namely, that surgeon's silk, untreated in any way, is apparently an aseptic material. The fact, however, is amply explained when one inquires into the processes to which it is subjected in the silk manufactories. As these processes[1] are of great interest, and have not, so far as I know, been previously noted in this connection, they may with advantage be detailed here.

The silk threads are "thrown" in China and Italy (chiefly in Milan), and come into this country coated with the dried viscid material of the cocoon, or, as it is technically called, *in the gum*. The processes to which these threads are subjected in English manufactories, have in view the removal of the *gum*, which is a valuable mordant, and the production of a thread as pure and white as possible. The *gum* is removed by boiling, and the threads are afterwards washed with the best curd soap. The soap is then removed by the process of "stoving," *i. e.* placing the threads for six to twelve hours in sulphur kilns. Finally, the threads are bleached by one or other of the patent bleaching powders. "Surgical" silk, which is the whitest silk obtainable, and more resistant than inferior qualities to the action of heat, is subjected twice to the process of "stoving." *Boiling*, *stoving*, and *bleaching* are, of course, powerful germicide processes, and, when it is remembered that after the silk has gone through three stages of preparation, the greatest care is taken to keep it free from dirt or dust by having manufactories in country districts,[2] by employing young girls, who are obliged to keep themselves neat and clean, and by immediate packing in clean cardboard boxes, we have ample explanation of the results of experiments 7, 8, and 10, 11, and, if I may venture to say so, of "*the use without evil consequences of silk ligatures*

[1] I am indebted to Mr. Gilles, of Messrs. Bayliss, Gilles, and Co., silk manufacturers, for a statement of these processes.

[2] Manufactories were first started in the country because it was difficult to obtain the silk perfectly white and pure in town atmospheres.

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which have not been subjected to any antiseptic preparation." I quote from Sir Joseph Lister's recent admirable address on the subject of antiseptic surgery,[1] in which, however, he states that until Metschnikoff's phagocyte theory cleared up the mystery, the fact, just noted, would have otherwise seemed to him incomprehensible.

With regard to sulphuro-chromic gut it is difficult to draw any definite conclusions from the results of experiments 1 to 6. Only one definite growth, however, was obtained in the nutrient broth, and that probably from a particle lightly adherent to the catgut, which, as has been noted, was not specially protected from dirt or dust. The haziness of the nutrient material, subsequent to the introduction of the gut, may possibly have been caused by the chemicals employed in the manufacture of sulphuro-chromic gut.

Plated wire was apparently aseptic. It is worthy of remark that in the pre-Listerian period wire had frequently been urged as the most satisfactory material for use, not only as sutures, but also as ligatures.[1]

SECOND SERIES.—*Preparations in "Antiseptic" Solutions.*

Under this head manufacturers' preparations of catgut and silk in aqueous and oily solutions of carbolic acid and mercuric chloride, and one preparation of silk in juniper oil, were tested. All the bottles in which the material was preserved had, with the exception of two, rubber or "gelatole" stoppers, through which the ends of the catgut or silk were threaded, so that the pieces could be drawn out and cut off without opening the

[1] "The present position of Antiseptic Surgery," 'Trans. Tenth Inter. Med. Cong.,' vol. i, p. 30. Also referred to by Prof. White in an article in the 'Annals of Surgery,' Jan., 1891, p. 3.

[2] Cf. Nunneley, "On the Substitution of Iron Wire for Thread and Silk as Ligatures for Arteries," 'Lancet,' 1862, i, p. 486.

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bottles. The two exceptions were the glass-stoppered and cork-stoppered bottles noted in the table below (Table II). The glass-stoppered bottle had in addition a parchment covering, and it was opened for the first time, since it had been obtained from the manufacturer, inside the inoculating box. The cork-stoppered bottle had been previously opened, pieces of the gut having been used for a major operation performed at the Royal Arsenal on the previous day. All the remaining bottles, except these two (which were obtained from army medical stores), were obtained direct from surgical instrument makers or manufacturers. The "gelatole"-stoppered bottles and the rubber-stoppered bottle of experiments 1 to 5 were prepared by foreign, the remainder by English firms. The silk in experiments 27, 28, as well as the bottle, grid and spools, had been boiled for one hour before the bottle was filled with the antiseptic solution. The catgut used in experiments 11 to 13 was the apparent cause of suppuration which took place in the case referred to above. Suppuration in the track of the stitches was almost invariably noted in many subsequent minor operations, in which this gut had to be used. I mention this here, because the occurrence of slight exudation of pus from the track of a suture, and from no other point, is in my experience quite common when carbolised catgut is used; and although it seems to indicate the impurity of the material, the attention of surgeons has not been sufficiently directed to its importance.

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TABLE II.—*Experiments with Material kept in "Antiseptic" Solutions.*

No. of experiment.	Nature of material and solution.	Where obtained.	Strength of solution.	Kind of stopper.	Date of experiment.	Result.
1	Catgut in "carbolised oil"	Surgical instrument maker	5%	Rubber	1890 Oct. 20	Permanently clear.
2	"	"	"	"	"	Haziness Oct. 24, 1890. Slight humidity Oct. 26, 1890. Slowly growing, non-liquefying growth on gelatine cultivations.
3	"	"	"	"	"	Haziness Oct. 24, 1890. Slight humidity Oct. 26, 1890. No growth on gelatine cultivations.
4	"	"	"	"	Oct. 29	Turbid Oct. 31, 1890. Rapid liquefying growths on gelatine cultivations.
5	"	"	"	"	"	Turbid Oct. 31, 1890. Rapid liquefying growths on gelatine cultivations.
6	"	Bottle in field medical pannier, Army Med. Stores, Woolwich	?	Glass	Oct. 24	Turbid and pedicle Oct. 29, 1890. *Staphylococcus cereus albus* cultivated on gelatine.
7	"	"	?	"	Oct. 29	Permanently clear.
8	"	"	?	"	"	Permanently clear.
9	"	"	?	"	"	Permanently clear.
10	"	"	?	"	"	Wrinkled pedicle Oct. 31, 1890. Very slow growth on gelatine cultivations, liquefying Feb. 13, 1891.
11	"	Bottle in use in Med. Dept., Royal Arsenal, obtained from surgical instrument maker	?	Cork	Nov. 12	Shreddy growth from thread Nov. 17, 1890. No turbidity.
12	"	"	?	"	"	Shreddy growth from thread Dec. 15, 1890. No turbidity.
13	"	"	?	"	"	Permanently clear.

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TABLE II (continued).

No. of experiment.	Nature of material and solution.	Where obtained.	Strength of solution.	Kind of stopper.	Date of experiment.	Result.
					1890	
14	Silk in "carbolised oil"	Manufacturer of antiseptic dressings	5%	"Gelatole"	Nov. 5	Turbid Nov. 8, 1890. Pedicle Nov. 10, 1890. Cultivation of *Staphylococci* and *Bacillus subtilis* on gelatine.
15	Catgut in oily solution of mercuric chloride	,,	1 in 2000	,,	Nov. 24	Permanently clear.
16	,,	,,	,,	,,	,,	Permanently clear.
17	Silk in oily solution of mercuric chloride	,,	,,	,,	Nov. 3	Growth from thread, turbidity and pedicle Nov. 10, 1890. Rapid liquefying growths on gelatine.
18	,,	,,	,,	,,	,,	Turbidity and pedicle Nov. 5, 1890. Rapid liquefying growths on gelatine.
19	,,	,,	,,	,,	Nov. 5	Growth from thread and turbidity Nov. 8, 1890. Rapid liquefying growths on gelatine.
20	"Chromicised" catgut in oil (juniper oil?)	,,	—	,,	Nov. 24	Clear Nov. 29, 1890. Tube accidently broken.
21	,,	,,	—	,,	,,	Permanently clear.
22	Silk in "juniper oil"	,,	—	,,	,,	Turbid Nov. 28, 1890.
23	,,	,,	—	,,	,,	Turbid and thick growth from thread after Dec. 8, 1890.
24	Silk in aqueous solution of carbolic acid	Surgical instrument maker	6%	Rubber	Oct. 24	Permanently clear.
25	,,	,,	,,	,,	Nov. 3	Permanently clear.
26	,,	,,	,,	,,	Nov. 5	Permanently clear.
27	Silk in aqueous solution of mercuric chloride	,,	1 in 10,000	,,	Nov. 28	Permanently clear.
28	,,	,,	,,	,,	,,	Permanently clear.

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These experiments show generally, I think, the untrustworthiness of manufacturer's preparations so far as *asepsis* is concerned. I cannot, of course, say under what conditions these preparations were made, and in the case of the "gelatole"-stoppered bottles it is possible that micro-organisms may have been obtained from the stopper itself. Most of the solutions, indeed I may say all the solutions, in which the material is stated to have been kept, are, by laboratory experiments, more or less antiseptic. So, at any rate, the next series of experiments would seem to prove. But it is a remarkable fact that Reverdin,[1] Thomson,[2] and Brunner,[3] in testing similar manufacturer's preparations, obtained similar evidence of septic impurities. Brunner, for example, tested 52 bottles obtained from 6 different manufactures. His results were briefly as follows:—*sublimate gut*, 25 bottles examined, no septic growths from any; *carbolised gut*, 12 bottles examined, no septic growths in 5, septic growths in 7; *chromicised gut*, 7 bottles examined, no septic growths in 3, septic growths in 4; *Ol. Juniperis gut*, 8 bottles examined, no septic growths in 5, septic growths in 3. Brunner states, therefore, with regard to Lister's process of preparing catgut with chromic acid, and Kocher's of preparing it with oil of juniper, that, "so far as these processes are carried out in manufactories, they afford no guarantee of the asepsis of the catgut." It must be noted that these observers tested catgut preparations only, and seemed inclined to blame the material, and not the solutions, for the septic results. It would appear, however, from the experiments in Table II that "antiseptic" preparations of silk are equally untrustworthy, and certainly afford no guarantee of the asepsis of the silk.

I do not for a moment wish to imply that manufacturers are not acting in strict good faith while pre-

[1] "Recherches sur la sterilisation du catgut et d'autres substances employées en chirurgie," 'Rev. Méd. de la Suisse romande,' 1888.

[2] "Experimentelle Untersuchungen über die gebraüchlichen Nahtmaterialen," &c., 'Centralb. f. Gynäk.,' 1889, No. 24. [3] Loc. cit.

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paring the material and the solutions, but I would direct attention to the fact that they may miss some essential point to which we have no clue, but which may make all the difference between an aseptic and a septic material, or between an antiseptic and an inert solution. An instance, occurring to myself, is instructive with reference to this very point. I asked a well-known surgical-instrument maker to prepare for me a rubber-stoppered bottle of silk, giving him written instructions regarding the method of preparing it aseptically. The silk was first directed to be rolled on glass spools, which were to be placed inside the bottle on a metal grid, and the free ends of the silk were to be threaded on needles. The bottle and its contents were then to be steamed for one hour in a steamer, and immediately afterwards the free ends of the silk were to be drawn through the rubber stopper and the bottle at once permanently closed. I gave these instructions in October, 1890, and obtained the bottle about a month afterwards. I then tested the silk in the laboratory, in the same way as I had tested the other preparations, with the following results :

TABLE IIIA.—*Experiments with Silk prepared "Aseptically" by Surgical-instrument Maker.*

No. of experiment.	Date of experiment.	Result.
1	Nov. 28, 1890	Permanently clear.
2	"	Pedicle on surface of fluid Dec. 1, 1890.
3	Dec. 1, 1890	Turbid on Dec. 3, 1890.
4	"	" "
5	"	Permanently clear.
6	"	" "

These results were, as may be imagined, somewhat disconcerting, three out of the six pieces of silk tested showing evidence of the presence of micro-organisms. I, consequently, applied to the manufacturer for a statement in writing, detailing the process to which the silk had actually been subjected. The following is the statement

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which I obtained :—"The bottles, grid, spools, needles, and silk were boiled for one hour at a temperature of 212° F. The silk was dried in a steam bath *after* the boiling process and *before* being put on the spools in the bottle." There was thus ample opportunity for the contamination of the silk, both by the hands of the workman and by the dust of the workshop, during the time intervening between the boiling process and the permanent closure of the bottle. Accordingly, I removed the rubber stopper in the laboratory, threaded the ends of the silk on needles, and placed the bottle and its contents in a steam steriliser for thirty-five minutes, the rubber stopper being in the interval placed in mercuric chloride solution (1 in 1000). Immediately on the removal of the bottle from the steriliser, the ends of the threads were drawn through the rubber stopper and the bottle permanently closed. I then tested the silk at different periods with the following results :

TABLE IIIB.—*Experiments with Silk prepared Aseptically in the Laboratory.*

No. of experiment.	Date of experiment.	Result.
1	Dec. 8, 1890	Permanently clear.
2	"	" "
3	"	" "
4	"	" "
5	Jan. 5, 1891	" "
6	"	" "
7	"	" "
8	"	" "
9	Jan. 13, 1891	" "
10	"	" "
11	"	" "
12	"	" "
13	"	" "
14	"	" "
15	"	" "
16	"	" "
17	"	" "
18	"	" "
19	"	" "
20	"	" "

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All the pieces of silk in the first thirteen of the experiments in Table IIIB were drawn out, cut off, and placed in the tubes by means of sterilised instruments, such as were employed in the previous experiments. In the remaining seven experiments the finger and thumb of the hand, previously washed with soap and water, dried, and then momentarily dipped in 1 in 1000 sublimate lotion, were employed instead of the forceps.

The contrast between the results of the two tables (Tables IIIA and IIIB) is sufficiently striking, and needs no further comment. The method of rendering and keeping silk aseptic, detailed in connection with the experiments of the latter table, seems a reliable and trustworthy method, if it is carried out strictly in accordance with the requirements of a bacteriological experiment. The process should not, therefore, be entrusted to manufacturers of antiseptic dressings or to surgical-instrument makers.

THIRD SERIES.—*The Antiseptic Value of the various Solutions in which Catgut and Silk are usually preserved.*

In these experiments surgeon's twisted silk was contaminated by being steeped for three hours in the meat infusion, rendered putrid by Experiment 14 of Table II, and containing growths of several micro-organisms, of which spore-bearing *Bacillus subtilis* and a form of *staphylococcus* (*Staphylococcus albus*) were differentiated. The silk, after being thus contaminated, was cut into pieces about half an inch long, dried under a bell-jar on clean blotting-paper for two days, and afterwards placed in the various solutions noted in Table IV. From time to time pieces were placed from the solutions in tubes of sterilised meat infusion, and incubated at 37° C., with the following results:

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TABLE IV.—*Showing results of placing Contaminated Silk in "Antiseptic" Solutions.*

Nature of the solution.	Length of time the threads remained in the solutions before being placed in the nutrient material, with the results.							
	30 seconds.	30 minutes.	1 hour.	24 hours.	3 days.	7 days.	39 days.	67 days.
1 in 2000 solution of mercuric chloride in distilled water	+	±	±	○	○	○	○	○
1 in 2000 solution of mercuric chloride in juniper oil	+	+	±	○	○	○	○	○
1 in 2000 solution of mercuric chloride in olive oil	+	+	+	+	±	±	⊙	○
1 in 20 solution of crystalline carbolic acid in distilled water	+	+	+	+	⊕	⊕	⊕	○
1 in 10 solution of crystalline carbolic acid in olive oil	+	+	+	+	+	+	+	○
Juniper oil (undiluted)	+	±	±	○	○	○	○	○
Inert control solution, containing ·6 % chloride of sodium in distilled water	+	+	+	+	+	+	+	+
Boiling water	?	○	?	?	?	?	?	?

EXPLANATIONS.

A cross (+) indicates that the nutrient meat infusion became turbid after 48 hours.

One line under the cross (±) indicates that the turbidity was delayed for 4 days, and *two lines* that it was delayed for 6 days.

A cross within a circle (⊕) indicates that all micro-organisms except one (*Bacillus subtilis*) ceased to grow.

A circle (○) indicates that no micro-organisms developed, and that the infusion remained permanently clear.

? indicates that no experiment was made under this particular head.

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It would appear from these results that all the solutions, with the exception of the inert control solution, have a germicide action varying considerably in degree. Employing gut prepared from rabbits which had died after inoculation with anthrax bacilli, Brunner[1] made similar experiments with the following results:—The gut was aseptic after remaining for 1½, 3, and 6 hours in 1 in 1000 aqueous solution of mercuric chloride, after 3 days in a 30 per cent., and after 12 days in a 20 per cent. solution of carbolic acid in oil, and after 36 hours in juniper oil. After 24 hours in juniper oil only some of the pieces tested were aseptic. The gut was also aseptic after exposure to dry heat for one hour according to Reverdin's method. I do not think that these results are quite satisfactory as a comparative test of the value of the solutions, because it is not certain that each piece of gut contained a similar number of or even any anthrax bacilli. However, if we take into consideration the fact that Brunner's solutions were double the strength of similar solutions of Table IV, the results of his experiments and mine bear a close resemblance to one another. With juniper oil there is a considerable discrepancy. The possibility of different qualities of juniper oil having been used may account for this.

How can we reconcile the difference between the results of Table II and the results of Table IV? a difference which I have already referred to, and which, as I have noted, Brunner and others have also observed; the difference, that is to say, between manufacturers' and laboratory preparations? There is no question of the material not having remained a sufficient time in the solutions. Attention has been already directed to the possibility of error on the part of the manufacturer. One other point, however, suggests itself. Is it not possible that the quantity of material is too great in proportion to the quantity of the solution in which it is kept? that, in other words, there is not sufficient antiseptic agent

[1] Loc. cit.

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present in the bottles to render the material aseptic throughout. Most of the bottles which I tested were of two ounce capacity, and the difference of weight, for example, between the bottle of silk in carbolised oil, of Experiment 14 of Table II, and the bottle of silk used in the experiments of Tables IIIA and IIIB, was one ounce, so that, roughly speaking, the bottle of Table II contained equal proportions of material and solution. Though no experimental tests were made to decide this question, it is possible that such a proportion is insufficient to destroy all the germs that may be present, not only, be it remembered, in the silk or catgut themselves, but also in the bottle, spools, and solvent medium, none of which probably undergo previous sterilisation. There seems, at any rate, a necessity for further investigation in this direction.

I am fully sensible of the fact that these experiments are incomplete from many points of view; but they are sufficient to teach one or two practical lessons.

1. All so-called "antiseptic" preparations must be received with considerable caution; so much so that those, who are not prepared to accept entirely Kocher's aphorism, "*Away with catgut,*" may with good reason consider the advisability of rejecting its so-called antiseptic preparations.

2. Asepsis can with certainty be obtained by the simplest, cheapest, and most efficient process of all, namely, by boiling or steaming.

3. Material thus rendered aseptic may be readily kept aseptic (by the method detailed in connection with the experiments of Table IIIB) in bottles containing no solutions of any kind. Such bottles have the further advantage of being at least half the weight of bottles containing a solution, an advantage of some importance, for example, in connection with the transport of field surgical equipment.

4. The preparations of such bottles cannot be entrusted to manufacturers.

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5. The methods of keeping ligature material aseptic by means of solutions, suggested from time to time by many eminent surgeons,[1] are unnecessary; but, should one of these methods be preferred, the most reliable solution to use is a solution of mercuric chloride in water (1 in 1000 or 1 in 2000).

6. The most aseptic material sold by surgical instrument makers is probably the pure undyed silk twist, untreated by any "antiseptic" method.

In concluding, I have gratefully to acknowledge my indebtedness to my friend, Dr. G. Sims Woodhead, the Director of the Laboratories, for his valuable supervision, and for the kind interest he took in the experiments, and to the Laboratories' Committee of the Conjoint Board in England for the liberal use I was permitted to make of the Laboratories.

[1] Brunner (loc. cit.) records the methods recommended from time to time by Lister, Macewen, Mickulicz, Kocher, Roux, Kümmel, Schede, Esmarch, Zweifel, Reverdin, and Beuchisser.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 74.)

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CASE

OF

TORSION OF THE SPERMATIC CORD,

WITH

STRANGULATION OF THE EPIDIDYMIS AND TESTICLE IN AN INCOMPLETELY DESCENDED ORGAN.

BY

THOMAS BRYANT, F.R.C.S., M.Ch.,

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Received December 12th, 1891—Read February 23rd, 1892.

On the evening of Thursday, September 26th, 1889, I was asked by Dr. Fox, of Barnet, to see P. G—, a boy æt. 15, with a swelling in his left inguinal and scrotal regions, and symptoms suggestive of strangulated hernia.

He was a healthy lad, but had suffered from constipation, and for five days had not had any action of the bowels. On Tuesday, the 24th, or two days before my visit, after a long bicycle run, unassociated with any accident, and to which he was accustomed, he went to bed as usual; but, quoting from a report of Dr. Fox's, "he woke his parents up during the night, complaining of pain in his scrotum and left groin." This was put down to the constipation, and a pill was administered to him next morning. After taking the pill he was sick and vomited. Dr. Fox was

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then sent for. He visited the boy about noon on the 25th, and found him flushed and hot, and suffering greatly from a pain in his left groin, shooting down into the scrotum, and some pain about his umbilicus.

On examining the painful parts, the left inguinal canal was clearly distended, and below the external ring there was a swelling which was supposed to be the left testicle; between the testicle and the external ring there was a small swelling, which, on pressure, seemed to be reducible, and gave, when pressed upon, a sensation, which Dr. Fox describes as somewhat similar to the crepitation caused by squeezing lung-tissue.

At 9 p.m. on the 25th, Dr. Ryle (Dr. Fox's partner) saw the boy with Dr. Fox, and, as the case presented some of the features of hernia, it was determined to apply the taxis very gently when the lad was under the influence of an anæsthetic. This was therefore done, but with no good effect. Later on the lower bowel was well emptied by means of an enema.

On the morning of the 26th the boy was found to have passed a bad night; his temperature was 101°, and all his local symptoms were increased. Under these circumstances Dr. Fox, still thinking that the case, though obscure, was one of hernia, asked me to see him.

I found the boy in bed, with a normal pulse and no fever. His abdomen was flaccid and not distended. The bowels had not acted since they had been well cleared out by an enema the day before. The left upper half of the scrotum, from the external abdominal ring, with the whole of the inguinal canal up to the internal abdominal ring was swollen. The swelling in the scrotum was somewhat flattened laterally, as in a case of epididymitis, and the whole of the inguinal canal was filled with a tense, dull, fluctuating swelling, in which there was no impulse on coughing; and the gentlest manipulation caused severe pain.

The diagnosis of the case was by no means clear; the local symptoms were consistent, to a degree, with those of hernia, but the general symptoms did not support this

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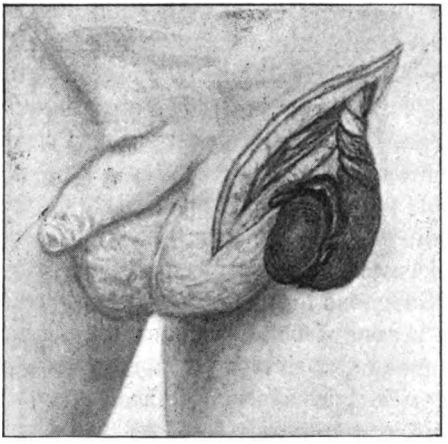
view. It was, however, thought right that an exploratory incision into the swelling should be made, and this I at once proceeded to do, as soon as the boy had been rendered insensible by chloroform. I made my incision from the external abdominal ring, and divided the soft parts down to the tunica vaginalis. Having done this a glistening coal-black object came into view through the sac, which, for the moment, was thought might be congested bowel. On opening the vaginal sac and laying open half the inguinal canal, much fluid, black blood escaped, and it became manifest that the black object which had been noticed was the testicle with its cord gorged with blood. On closer examination, it was then found that the cord had been twisted upon its axis in the inguinal canal, and that, as a result, the testis had become strangu-



lated with the epididymis. Three half twists made inwards completely untwisted the cord, which, when this had been done, looked like a bloated leech attached to a testicle, black from venous congestion or strangulation. As the testicle was warm, and therefore viable, it was deemed right to give it a chance of recovery; it was therefore replaced in its vaginal sac, and the soft parts

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were sutured together, after having been well washed with iodine water, and dusted with iodol and boracic acid (1 part to 4). The wound was dressed with blue wool.

After the operation all symptoms at once disappeared, and the wound healed by quick union. For several weeks subsequently the testicle and cord remained thickened; but this soon disappeared, and later on the testicle atrophied. The boy is now quite well.

I should add that in this case the left testicle had only just passed through the external abdominal ring, and that to expose the cord where it was twisted, I had not only to lay open the sac of the tunica vaginalis, but the inner half of the inguinal canal. The internal abdominal ring seemed to be quite closed.

Remarks.—When this case first occurred to me, I thought it unique, as I was unable to find in any work a reference to one similar, although I am disposed to think that in Mr. Jacobson's admirable paper on Diseases of the Testicle, published in the 3rd vol., 3rd edition, of Mr. Holmes's 'Surgery,' p. 471, an example, in a lad aged nineteen, is recorded under another heading.

In the summer of this year, however, a preparation, consisting of a testicle and its cord, was forwarded by Mr. G. Nash, the then house-surgeon to the South Devon and East Cornwall Hospital, to the museum of the Royal College of Surgeons for examination, which was evidently taken from a case of the same nature. I have thought it wise, therefore, to introduce a report of this case in this paper, with a description of the preparation from the pathological curator of our College, although it has been published in one of the weekly journals ('British Med. Journal,' June 6th, 1891) by Mr. G. Nash.

A. H—, a boy æt. 16, was admitted on January 21st, 1891, at 4.45 p.m., into the South Devon and East Cornwall Hospital, Plymouth, under the care of Mr. C. Whipple.

He stated that for seven or eight years he had noticed

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a lump in his left groin, which occasionally disappeared within his abdomen. This he believed to be his left testicle. The day before his admission, at 4 p.m., he strained himself, and felt something give way in the left groin. At 2 a.m., on the 21st, he noticed a lump in the groin, and at 8 a.m. began to vomit. He consulted a medical man, who diagnosed a strangulated hernia, and sent him to the hospital with a note to say he had stercoraceous vomiting. Bowels acted slightly at 2 p.m. that day; before that two days ago.

On examination.—In the left groin was an hour-glass-shaped swelling. The lower half of this was the left testicle, lying in the upper part of the scrotum. The upper, lying on the external abdominal ring, about the size of a hen's egg, was very tense, quite dull on percussion, and gave no impulse on coughing. The pubes was shaved and washed; at 5.15 p.m. methylene was given. Mr. Whipple made an incision over the upper swelling, and a dark-coloured tense sac was exposed. This was opened and some blood-stained fluid gushed out. A dark claret-coloured coiled mass presented, and after careful examination, this was made out to be a much enlarged, strangulated epididymis. Lying in the lower half of the sac was the body of the testis, of about normal size; and attached to it, and forming a circle round its hilum, was a band of omentum. This tag was torn through, and the proximal part of the omentum, which was unaltered in appearance, was returned within the abdomen through a large inguinal canal. The epididymis appeared to be twisted twice upon its own axis. This was pulled down and untwisted. The epididymis was ligatured as high up as possible, and it and the testis were removed. The pedicle was dropped back. The sac was ligatured in halves and cut away, and the pillar of the external ring sutured. A rapid recovery ensued.

The specimen, A4262, Museum R.C.S. (shown), was examined by Mr. J. H. Targett, of the Royal College of Surgeons, and reported upon as follows:

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"The preparation consists of the body and epididymis of the left testis, and the adjacent portion of the mesorchium. The body of the testis is normal. Between it and the globus major there is a deep groove, at the bottom of which lies a loop of omentum, which completely, but not tightly, encircles the attachment of the body of the testis to the epididymis. The torn end of this omentum is marked, the mesorchium measures nearly two inches across, and is irregularly swollen on either side from distension of the vessels and extravasation of blood between the layers of the peritoneum. The position of the vas deferens is indicated. At the junction of the mesorchium and epididymis, in the position of the globus minor, there is a well-marked constriction, which appears to have resulted from a severe twisting of the epididymis upon the mesorchium. To this torsion the acute strangulation of the epididymis was probably due, as well as the hæmorrhage into the fatty tissue of the mesorchium.

"On re-examining this specimen, I can see no other explanation than that the epididymis, or, to speak more correctly, the mesorchium, has become twisted as an ovarian pedicle may do, and so the circulation has become obstructed; for there is a well-marked groove at the base of the epididymis, where, I presume, the twisting has taken place. I have not met," writes Mr. Targett, "with a similar complicated, strangulated, undescended testicle, nor do I know of any case recorded in the ordinary text-books."

Remarks.—In both of these cases to which your attention has just been drawn, the twisting of the cord took place in boys; in both the condition affected the left side, and in both there was an incomplete descent of the affected testicle.

In my own case the torsion took place within the inguinal canal. In Mr. Nash's case it was said to have occurred at the external abdominal ring. In my own case the strangulation of the parts had passed several stages beyond Mr. Nash's.

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By what process the torsion of the cord was brought about I am profoundly ignorant, for neither accident, nor any of the changes which take place in the testicle during its physiological descent into the scrotum, seem capable of helping us towards a solution of the problem. I leave this point, therefore, for future solution.

I am, however, fully certain that these cases have occurred before, and have been overlooked, for if neither of the two cases I have brought before you had been operated upon, their true nature would never have been made known. Mechanical violence in the form of taxis would probably have been applied under the mistaken notion that the case was one of hernia, when, as a result, suppuration or gangrene of the injured organ might have ensued, and the case would eventually have been described as one of inflammation, with suppuration or gangrene of an imperfectly descended organ. On the other hand, when these serious results did not take place, and atrophy of the gland was the result of the local action, the case would have been recorded as one of atrophy after inflammation.

With these few remarks I leave the case for your consideration, and trust the Fellows of this Society will deem it worthy of a place in the records of surgery.

Ectopic Testicle, with a very short Pedicle, in the inguinal canal, combined with Inguinal Hernia, twisting of the Pedicle, with Hæmatoma of the Epididymis, and beginning gangrene, discovered by operation; pyogenic Cocci found; recovery. By W. W. KEEN, Professor of the Principles of Surgery, and of Clinical Surgery in the Jefferson Medical College, Philadelphia, Pa.

R. Van S—, æt. 23, was admitted to the Jefferson Medical College Hospital, February 17th, 1891. The following history was elicited:—From birth he had been troubled with a right, oblique, reducible inguinal hernia, and also an undescended testicle on the same side.

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Three days before his admission he had an attack of vomiting, which ceased in the course of the day, and did not recur. The next day the bowels were constipated, and he observed an increase in the size of the tumour, which he attributed to the descent of the hernia. He attempted to reduce this, but failed. Considerable pain followed the attempt, and the tumour became exceedingly tender. Physical examination showed a tumour in the right inguinal canal, which was in part tympanitic, and in part very solid and resisting. It was so tender that no manipulation could be made with a view to taxis, excepting under an anæsthetic. The skin over the tumour also was beginning to be markedly inflamed.

My diagnosis was a combined hernia and ectopic testicle, as was evident from the absence of the right testicle in the scrotum. Accordingly, I operated on him the next day in the clinic.

Before making an incision, my colleague, Professor Brinton, while examining the tumour, was able very readily to reduce the hernia by taxis. An incision was then made over the tumour in the axis of the inguinal canal. The moment that I reached the testicle, I found a curious state of affairs, such as I was totally unfamiliar with. I found that the testicle lay in the inguinal canal, anchored by a pedicle about an inch long, which was attached to the abdominal wall at the internal inguinal ring. The testicle had been rotated three half-turns, and this had so obstructed the vessels, that a hæmatoma had formed just behind the testicle, the hæmatoma being about the same size as the testis itself. The hæmatoma was soft and black. Around the testicle and the hæmatoma was a small amount of fluid, with a slightly-marked fæcal odour. The internal ring was patulous and easily admitted the thumb.

I ligated the twisted cord, removed the testicle and cord, and then sutured the walls of the inguinal canal and of the internal ring together, after most careful and thorough disinfection and provision for drainage, as it

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was probable that, in spite of the disinfection of the parts, suppuration would follow. Slight suppuration took place, with a moderate rise of temperature, and the wound finally healed by second intention throughout its length, giving a sound cicatrix, without any return of the hernia. In December, 1891, when the patient was seen, he was quite well and cured of his hernia.

The tumour was examined by Dr. W. M. L. Coplin and Dr. David Bevan, Bacteriological Assistants in the clinic. The latter made most careful cultures and microscopic sections, and furnished the following pathological report:

"In tubes inoculated from the serum about the testicle, there developed in twenty-four hours, at a temperature of 97° F., a well-marked growth which, however, then ceased to grow further. In appearance it was dull grey and opaque. From this growth a peculiar and offensive odour emanated. Microscopic examination determined the growth to consist of cocci, which in diameter measure ·8μ, arranged in zooglea, and of bacilli, short and slender, measuring in length about 1·5μ, and in breadth ·6μ. These organisms stain well with aniline dyes and by Gram's method.

"The hæmatoma was in an advanced condition of gangrene, and the process was evidently extending quite rapidly to the testicle, itself, showing in different parts various stages of disintegration. The vessels were thrombosed and many of them ruptured. Blood-corpuscles were diffused throughout the testicle. No micro-organisms were observed in the testicle itself."

Remarks.—The condition here reported is an extremely rare one, if I may judge from the fact that in thirty years of active hospital life I have never seen anything like it, and, moreover, none of my surgical friends of large experience have either seen or heard of a similar case, nor do I find any reference to it in the text-books.

Last September, when Mr. Bryant was in this country, I related the case to him; and as he had had a similar

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case, and also believed it to be very rare, at his request I have furnished him with an account of the case, with a view to its publication with his.

Undoubtedly, the efforts of the patient to reduce the supposed hernia were the cause of the rotation of the testicle, followed by the production of the hæmatoma and the rapidly increasing gangrene, beginning in the hæmatoma, and extending to the testicle itself. The infection by the two forms of bacteria is a matter of interest also, especially as there was a hernia combined with the ectopic testicle, and there is a strong presumption that the bacteria obtained access to the hæmatoma from the bowel itself. The use of the bichloride during the operation probably interfered with the development of the bacteria in the cultures. It could not, therefore, be determined whether the bacillus was the bacillus colicommunis or not, but this was probably the fact.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 80.)

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CASE

OF

TORSION OF THE SPERMATIC CORD IN A DOG.

BY

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ASSISTANT SURGEON TO THE MIDDLESEX HOSPITAL.

Received and read February 23rd, 1893.

I HAVE recently had an opportunity of studying an example of axial rotation of a retained testis, which was, in some respects, even more exceptional than the interesting cases brought under the notice of the Society by Mr. Bryant. The patient was an old pug-dog which died somewhat obscurely, and at the post-mortem examination the genital organs were removed and forwarded to Professor Penberthy, who, knowing that I was particularly interested in the question of axial rotation of abdominal viscera and tumours, was so generous as to place the organs in my hands for examination and preservation.

The specimen consists of the bladder, prostate, part of the penis, and the testicles. The right testicle was retained and enormously enlarged in consequence of a new-growth which had formed in its body. The

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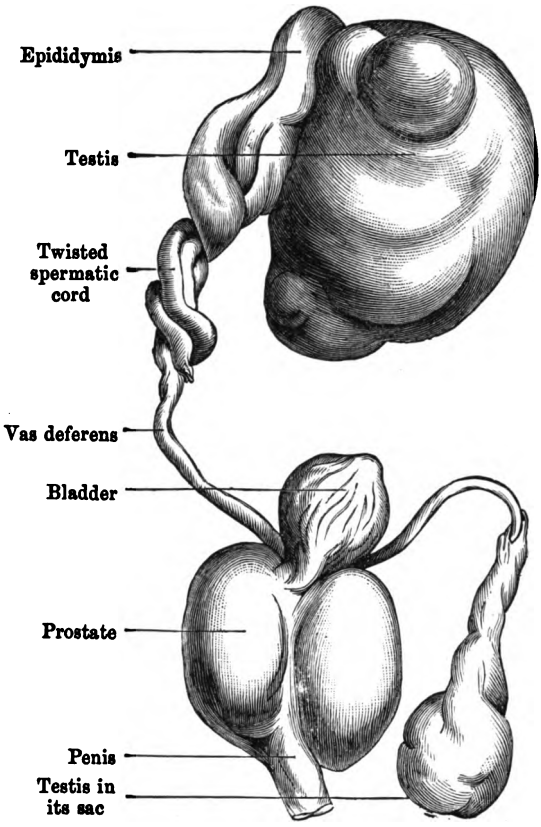
testicle, which was in close relation with the kidney, had rotated on its long axis two and a half times. This movement had twisted the cord acutely, and led to congestion of the tumour and epididymis. The appearance



Tumour of a retained testis in a pug-dog. The testis had rotated on its long axis two and a half times.

of these parts was identical with that of an ovarian cyst which had undergone rotation. In the case of the pug, portions of the peri-renal fat had been implicated in the twisted portion of the spermatic cord.

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This unusual condition induced me to search for similar cases. Dr. Bramann has contributed to Langenbeck's Archiv., Bd. 40, S. 137, an interesting paper concerning retained testes, in which he refers to rotation of the testis, and mentions that it may be so acute as to lead to necrosis of the gland. In support of this statement, Bramann gives abstracts of two cases described by Nicoladoni, and gives the reference to another reported by Cohen. One of the cases reported by Nicoladoni is very similar to the cases, the subject of Mr. Bryant's very valuable paper. As far as can be ascertained, the pug's case is exceptional, as the testicle which rotated was occupied by a tumour. Retained testes in dogs, bulls, horses, and rams are as liable as retained testes in men to become the seat of malignant disease.

We have now evidence that nearly every viscus in the belly is liable to rotate axially and twist its pedicle, *e. g.* testicles, kidneys, spleen, ovaries, and the gravid uterus. Tumours, such as ovarian cysts, dilated tubes, uterine myomata, omental hydatids, and the like are also liable to rotation.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 80.)

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THE OPERATIVE TREATMENT

OF

CONGENITAL DISLOCATION OF THE HIP.

BY

RICHARD BARWELL,

CONSULTING SURGEON, CHARING CROSS HOSPITAL.

Received December 15th, 1891—Read March 22nd, 1892.

The term "congenital dislocation of the hip" is somewhat misleading, for the anatomical condition is not a displacement of a normal head of the femur out of a perfect acetabulum, but a greater or less truncation of the former and an absence more or less complete of the latter.[1] A more minute description of these osseous defects does not enter into my present purpose; nor need I here discuss the well-known symptoms of this condition —the breadth of the buttocks—the waddling, uncertain and balancing gait—the mobility, restricted in certain ways, excessive in others and in false directions. Yet there are one or two points in the semeiology of the deformity having important bearings on its diagnosis and

[1] Excellent specimens were shown and described by Messrs. Lockwood, Bowlby, Morgan, Adams, Power, and Shattock, 'Pathological Transactions,' vol. xxxviii, p. 780.

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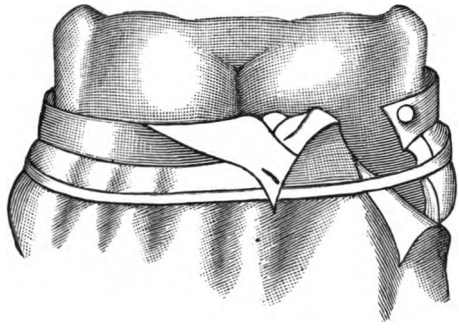
prognosis, certain of which have not hitherto been observed, while others have not been sufficiently emphasized. In the former category is the following very characteristic symptom.

The patient stands erect; the surgeon, seated directly behind her, causes her to keep the knees straight and to bow forwards, as though to touch the instep with the finger-tips. When by this means the trunk, more especially the pelvis, is brought to be almost or quite horizontal on the perpendicular thighs, the trochanters, or if the deformity be unilateral the one trochanter,[1] will project upward from the back of the pelvis in a very remarkable manner, which baffles my powers of description. Therefore I must beg the Fellows to excuse the inartistic



crudeness of an outline sketch which I took in 1890 of a child that was sent to me by Dr. Sprott, of Beeston. However rough, the sketch shows quite characteristically and in no degree exaggerated the upward and backward prominence of the trochanters, which is the more valuable as a diagnostic sign because it is never found in any

[1] In the unilateral deformity this conformation often does not appear spontaneously, because the child puts no weight on the dislocated limb. In such circumstances the surgeon, placing his hand on the sound side of the pelvis, gently pushes that part over towards the deformed side; as soon as its full share of weight falls on the dislocated limb the characteristic projection shows itself.

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other condition of the joint, and is, in my experience, always present in so-called congenital dislocation.

Another point is the ease with which, when the patient lies supine, the surgeon can shorten or lengthen the limb. He can, immobilising the pelvis with one hand, push the thigh up till the trochanter lies very nearly on a level with the iliac crest; then by traction he can cause it to descend until it lies at, or in some cases a little below, its proper level. It is important to observe whether or no, when drawing the limb down, there occur just as the trochanter reaches its proper place a certain jolt or jog. If it do occur the surgeon should gently push the bone up again, noting if at the same level a like jolt be felt. If such do take place he should observe, whether it be more marked and more abrupt than that which is produced by downward movement. The sign of course indicates that the side of the pelvis is not quite an even surface; but that there is some sort of ridge at the locality normally occupied by the upper lip of the acetabulum: the ridge may not necessarily be formed by a projection, but at least by the edge of a depression.[1] Those cases, which have this symptom most fully marked, are, *cæteris paribus*, most promising for treatment.

The actual amount of abnormal upward and downward movement of the femur upon the pelvis, and the amount of backward projection in the stooping posture above described, are in accordance, not with the conformation of either bone, but with the greater or less laxity of the capsular ligaments and muscles; hence such mobility is always greater in those children who have already walked for some time.

Before passing on to describe the form of treatment, which I have adopted for some eight years I must ask for a little patience for a short *résumé* of the principal methods hitherto made known.

[1] As far as I know, Buckminster Brown is the only surgeon who has taken notice of this "jog," and he did not observe it till the case had been some weeks under treatment.

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1. *Division of rotator and capsular muscles.*—Guérin speaks of these muscles as obstacles to reduction, he divided them pretty freely, especially all three glutei. His sections appear to have also involved a considerable part of the capsular ligament; likewise he scarified the bone in the neighbourhood of the acetabulum. The report of a Commission (1848) is not favorable.[1] Mr. Brodhurst advises free division of the two smaller glutei and of the rotator and capsular muscles, saying that when these "are freely divided a thigh splint and groin pad effectually prevent further displacement."[2]

2. *Prolonged rest with extension.*—This method originated with Pravaz, of Lyons, in 1838, and was vigorously defended by his pupil, Gillebert d'Hercourt,[3] against the scepticism of Bouvier. Difficult as it may be to decide on the merits of an old controversy, a certain measure of success must be awarded to Pravaz. Carnochau's well-known memoir in 1850 advocates a similar plan, to be carried out by means of very complicated machinery.[4] I do not find in his book reference to any case treated by him after this method. In 1882 Dr. Buckminster Brown began the treatment of a girl, aged four, by a like method, but with different apparatus, and after twenty months effected a relative cure; the child being able to walk, and the weakness being such as might probably be removed by exercise.[5]

Mr. Wm. Adams has in this country carried out and zealously advocated this method. He refers to six cases, and reports on two which were unilateral; one was kept in recumbency for "the full period of two years, the other

[1] The Commission specially appointed reported on five cases at the end of two years. One had been attacked with zymotic disease, and was untouched; two very slightly, if at all, benefited; two still under treatment, and not in a promising condition.

[2] Holmes's 'System of Surgery,' vol. iii, p. 800.

[3] 'De la Curabilité des Luxations congénitales fémorales,' Lyon, 1854.

[4] 'On the Etiology, Pathology, and Treatment of Congenital Dislocations of the Head of the Femur,' New York, 1854.

[5] 'Double Congenital Displacement of the Hip,' New York, 1885.

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for two years and seven months; the ages were two years, and one year and nine months respectively." After cessation of extension the use of a special steel instrument and crutches was strictly enforced; the patients do not appear to have dispensed with these appliances on the date of the last reports, three years after the commencement of treatment, but the prospect was satisfactory—the limbs were slightly shortened. Mr. Adams does not say if the other four cases were treated, or if they were failures of treatment.[1]

Thus it must be conceded—indeed, it has been proved by Pravaz, by Buckminster Brown, and William Adams—that entire recumbency for two or for two and a half years, followed by another year or year and a half of instruments and crutches, may, in a certain proportion of cases, be followed by a gratifying amount of success, if such treatment be undertaken at a very early age. Indeed, Mr. Adams considers this an essential element of success, for he says that "at a later period, *i. e.* after ten years of age, there cannot be any reasonable hope of much improvement."

I would now submit that this prolonged treatment—doubtless very deterrent to parents—may be greatly shortened, and the ultimate success rendered more sure, even in much older patients, by the division of certain muscles, but decidedly not of the rotator and capsular muscles; my contention being that M. Guérin did not divide the right ones, and that the statements of Mr. Brodhurst appear irreconcilable with pathological anatomy and with the consensus of clinical experience.

I was led to the view on which my practice is founded by observations made on a child three and a half years old with right congenital dislocation. They were the more easily verified because the girl was very thin. I observed that attempts to draw the femur down were strongly resisted by the adductors, which became more

[1] Paper read at Annual Meeting of British Medical Association at Leeds August, 1889.

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and more tense as the traction increased. A time was fixed some three weeks later for the commencement of treatment; but the child was taken to the sea-side, contracted some form of contagious sore throat, and died.

About two years after occurred the following case:

October 13th, 1883. Miss T—, æt. 11, was born easily and naturally, and till lately has lived in South America. When a long time beyond the proper age for walking it was observed that she could support hardly any weight on the lower limbs. She has all her life been scarcely able to walk at all, but the cause of this inability was not elucidated until the above date, when Dr. Robertson, of Dulwich, who had been consulted, brought the child to me. The signs of double congenital dislocation were all markedly present—both thighs could be pushed up and drawn down very readily, the left one further in both directions than the right; in both the jolt already mentioned was distinctly felt just as the trochanter reached the normal level. On both sides, when the limbs were drawn down, the adductors became very tense. In the erect posture, which she could only maintain by grasping some piece of furniture, the pelvis was almost horizontal, lumbar lordosis being therefore excessive. There was no possibility of doubt as to the nature of the deformity.

October 23rd I operated, first on the left side by dividing the two lesser glutei through two skin punctures, one a little in front of, the other some distance behind, the great trochanter. I then divided the adductors. In performing this operation it is essential to avoid cutting through the bodies of the muscles, otherwise some subcutaneous bleeding and ecchymosis result; therefore it is necessary to keep the knife quite close to the pelvic bones.

Thus Dr. Robertson drew the thigh very slightly outwards, while I, finding the tendon of the adductor longus, passed in my tenotome just at its outer border quite close to the pubes, with the cutting edge upward, which I carried gently up until I felt the bone; then, turning the

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edge inward, peeled that tendon from its origin, rather than simply divided it. In the same way, and while Dr. Robertson was still further abducting the thigh, the anterior fibres of the adductor magnus, the whole of the adductor brevis, and the gracilis were divided. I now felt with my left forefinger the condition above the inner condyle of the strong inner part of the great adductor; and as, when the thigh was abducted and at the same time drawn well downward, that tendon was in strong tension, I glided the tenotome still deeper, using my left index placed on the tuber ischii as a guide, and divided the origin of that part of the muscle; also, through the puncture for dividing the gluteus minimus, a strong band of fascia that tightened excessively on downward traction was cut. Through the posterior gluteal puncture a strong tenotome, with a long blunt and a short cutting portion, was passed to the place of the ridge, if I may so call it, which represents the upper lip of the acetabulum. Here I endeavoured, and, I believe, succeeded in turning up a flap of periosteum about one and a half inches long by half an inch wide. Pieces of plaster were put over the little orifices and covered with collodion.

A precisely similar operation, except that it was unnecessary to loosen the posterior part of the adductor magnus, was then performed on the right side; but when the thigh was drawn down the knee became rigid and could not be completely flexed, while endeavours to do so tightened the tendon of origin of the rectus. A tenotome was slipped under it, the edge turned forward, so that when Dr. Robertson again tried to bend the knee the tendon parted. The limbs could now with very slight force be drawn down to or below their proper level, and on cessation of traction showed no inclination to ride up again. The lordosis was, I thought, slightly diminished; but the punctures being well protected, I turned the child on the side, and stooping behind her, placing one hand across the sacrum and grasping the knee with the other, I hyperextended the thighs one after the other as far as

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I could with the object of stretching the ileo-psoas. The lordosis was now decidedly less.

A firm belt, which had previously been carefully fitted, and was provided with under-straps, was tightly secured round the pelvis; the child was kept in dorsal decubitus with the limbs somewhat abducted, and weight extension was applied.

Neither pain, pyrexia, nor ecchymosis followed the operation. For the next two months Dr. Robertson looked after her with great skill, I only seing her occasionally. At the end of that period, viz. exactly nine weeks after the operation, I found any attempt to press the thigh upwards on the pelvis by manual force was pretty firmly resisted. She was provided with a three-wheeled go-cart with crutch handles (a special appliance), and with instruments preventing weight falling on the hip-joints, and she was strictly enjoined not to walk even a single step without both these mechanisms.

In October, 1884, I allowed her to stand a few minutes in my presence, and found the hip-joints very firm. I tested her, whilst recumbent, with Nelaton's line, and found that even when Dr. Robertson was pressing the thigh with some little force upward, the trochanter of each side was hardly at all above the proper level. I advised her to go on with the hip instrument; the go-cart was discarded, but she was supplied with crutches. A donkey with large saddle for riding about the roads was provided.

July, 1885.—She walked very well, with barely a suspicion of a balancing or swaying movement. There was still slight, but only slight, lumbar lordosis.

November, 1891.—I do not think I saw her after the above date, but I heard of her in various parts of England. In November of this year I wrote, asking her to describe her condition, and suggesting certain points for her greater attention in her reply. I subjoin those parts of her letter which have reference to her physical condition.

"I never feel any pain in my hips at all. I seem to myself to limp a little, but a great many people, who have

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known me for months, have not noticed any limp at all, and, unless I tell them, have no idea that there has been anything wrong with me. . . . The loins are a little bent forward, and I am broad across the hips, but I have large bones naturally. I can walk four or five miles without being at all tired; sometimes I walk more, and I do not mind stairs. I walk very well uphill; in doing so I prefer (though I can do quite well without it) to have an umbrella for a walking-stick. Yesterday I walked up Oliver's Mount (a steep hill near Scarborough). In Scilly (this in 1889 and 1890) I used to climb about the rocks famously, and go out boating whenever the weather was fine, without difficulty in getting in and out of the boats. I made myself quite famous by going out to the Bishop's Rock Lighthouse with another lady and father, being drawn up by a rope seventy feet, and let down again into our boat in the same manner, after which we had to scramble into a steamer. Another day we visited an old ruined castle, and I got to the top of it by granite steps very irregular, and without any rails to hold on to, which is more than some, even gentlemen, of the party would attempt."[1]

Case 2 (May 8th, 1886).—Master C—, æt. 7 years and 2 months, was born naturally in Australia, but has always limped, which his mother was told arose from a limb being

[1] Since this paper was read at the Society Miss T— called upon and kindly gave me the opportunity of a thorough examination. She is now just over twenty years of age, not tall, but otherwise a large, powerful-framed young woman, weighing probably between eleven and twelve stone. Her walk in indoor costume is perfect, with no more sway of the figure than is ordinarily possessed by women with large pelvic development. The garments being sufficiently removed, it is seen that the breadth of the hips, mentioned in her letter, is entirely due to the fact that the pelvis is large and broad. There may be, but this is doubtful, a little more lordosis than is usual with women of that stature. In causing her to walk, I, following with my palms upon the trochanters, found that they did not rise on the pelvis as she put the weight on either foot, their play seemed normal; but at the right hip I felt a slight click when the limb of that side was behind, and just before the other foot came to the ground. She could, by separating the feet, abduct

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naturally short, and that he must wear a high shoe. He suffered no pain, was very lively and full of play—rather too much so. I found the limb in dorsal recumbency an inch and a half short, but it could be pushed still further up so as to be two and a quarter inches short, and could be drawn down to be only about three eighths of an inch short; and this latter seemed to depend on absolute loss of length, not from position of the femur. In thus drawing the limb down a very distinct jog was felt. There was a good deal of lordosis, and some lateral twist of the lumbar spine. The sway of the body in walking was very considerable, and there was great instability while endeavouring to stand on the left limb. I gave my opinion, and at the request of the family was asked to meet Sir J. Paget. The consultation took place on May 12th. Still another opinion was desired, and Mr. Erichsen met me on the 17th, but there was in reality no doubt as to the nature of the case.

May 22nd.—I divided the two smaller glutei, and the adductors, and found myself also compelled to divide the rectus. Weight extension in the recumbent position was employed, and immobility was obtained by means of sandbags. No pyrexia, ecchymosis, nor pain followed the operation.

I attended to the boy as occasion required, but found that he kicked and moved about a great deal too much, in spite of all the arrangements that were possible. His brothers and sisters, too, augmented his too lively spirits; nevertheless, by a firm pelvic band fastened to the bedstead and considerable weight extension, I believe I kept the hip in fair position.

July 7th.—I allowed him to get up, and with the same steel instrument and crutches as in Case 1 to go about

the limbs like other women, and (a severer test) she could stand with the legs crossed, leaving an interval of four and a half inches between the outer malleoli. Recumbent, the limbs were capable of all movements. My strongest efforts to force the femur upward on the pelvis made no difference in the position of the trochanter.

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on one floor and out of doors in the carriage. There was very considerable resistance in pushing up the hip.

August 2nd.—The boy walked, with the instrument on, very much better. He may now do so for half an hour without crutches. At the above date he went with the rest of the family to Scotland, and with strict directions from me to continue in the same way.

October 1st.—I fear my directions have not been very well obeyed; the boy wearing the instrument walks well, but with some sway of the body. There is good resistance in endeavouring to push the thigh upward, but the trochanter now lies above Nelaton's line, which was not the case before he left London.

18th.—At this date he was taken back to the colony and ordered to continue the instrument, in which he walks well, but he rushes about wildly.

Case 3 (January 3rd, 1892).—Miss Muriel R—, æt. 2½ years, was sent to me at above date by Dr. Belcher Cooke. The birth was difficult but natural, without instruments. At the end of the month Mrs. R— undertook the management of her child, and observed something wrong, a lump about the hip-joint, which her then medical attendant said was nothing. After some weeks, the family having removed, Dr. Cooke saw the child and found the limb very feeble and almost without sensation. Under treatment these troubles diminished. At ten months of age she was taken to the Orthopædic Hospital, where, according to Mrs. R—, the case was diagnosed as an old fracture of the thigh; she was told that the child would always be lame, and would have to wear a high shoe.

When, at the above date, the child was brought to me she could bear hardly any weight on the left leg. When holding her by the axillæ and shoulders and directing her to lift the right foot, she instantly put it down again. Without some extraneous support she could not stand on the left limb at all—in fact, could not be induced to lift the right foot. Lordosis and a curve of the lumbar spine

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to the left were very marked when she stood with both feet close together.

Placed lying on the back, the left limb was found to be 1¼ inches shorter than the right, the great trochanter lying considerably above Nelaton's line; but if drawn down so that that point of bone more nearly approached that line, the limb was ⅝ inch short. By upward pressure, any movement of the pelvis itself being guarded against, the trochanter could be made to glide upward, and to lie much above Nelaton's line, very near to the crest of the ilium, and then the limb was 2⅜ inches short. When she, with straight knees, bows forward, upward projection of the trochanter was well marked. This mobility of the trochanter on the pelvis and its projection in stooping left no room to doubt the nature of the case. The jog on drawing the limb either down or up could only be felt when it was abducted and rotated outward—a condition which indicated, as I believed, a small head of the femur.

January 8th.—I divided both the lesser glutei and the adductors—and after full examination, also the rectus—stretched the ilio-psoas and turned back the periosteum or perichondrium, for I found parts at edge of acetabulum quite soft. The same method of immobilising and extending the limb was adopted. In exactly a month she left the private hospital, with the steel extension instrument applied and with crutches. She was to walk at first only for a few minutes at a time, and not oftener than four times a day; the instrument was to be applied before she was allowed to rise, and as soon as the weight extension was removed—the change back to weight extension was to be effected.

In April of the same year she was allowed to discard the crutches, and in November, as the hip resisted strongly manual attempts to push the thigh upwards, weight extension was discontinued.

May, 1891.—The child appeared to be very strong on the left leg. I tested her carefully, and did not find the trochanter rise as she put weight on that foot. She was

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to be allowed to walk for five minutes twice in the day without any appliance.

August.—The child is virtually well. The condition is as follows:—The trochanter lies just a shade above Nelaton's line. The left leg is not quite ¼ inch shorter than the right—this appears due to want of growth in the femur. She walks very well; the very slight limp quite disappears when a thickness of ⅛ inch is added to the shoe. The joint, or what does duty for a joint, at the hip is quite firm. There is no lumbar lordosis.[1]

I would now beg the Fellows of this Society to consider the action of muscles in upward congenital dislocation of the femur—by far the most usual form. It must be evident that the muscles, which pass from the pelvis outward and across the axis of the thigh to be inserted into the neighbourhood of the trochanter, have, among other actions, that of keeping the femur close to the innominate bone—of reinforcing the capsule; and that, therefore, their integrity is most important, especially if their course, like that of the obturators, be also a little upward.

Those muscles which, arising from the pelvis, pass downward to their insertion in the femur, more or less parallel to the axis of the thigh, must, in the absence of an acetabulum, tend to draw the head of the bone upward, to reproduce the original displacement. In fact, the aim and object of extension is to counteract this tendency; but while those muscles are entire, it takes a long time—at least two years according to Mr. Adams—to overcome their displacing activity. Hence in his case Dr. Buckminster Brown found the dislocation recurred every morning, although he put the thigh into position every night, and this continued for three weeks.

All this great loss of time, weariness, and risk to health—all this excessive tendency to recurrence may be overcome by the safe and painless operation above described;

[1] The mother of this patient kindly brought her to the meeting of the Society, when the above statements were verified by the Fellows attending.

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moreover patients, the special cause of whose lameness and deformity has not been diagnosed till rather a late period—that is to say, patients whom there is no reasonable hope of benefiting by extension and recumbency—can be rendered as active and agile as my Case 1.

More than this, while those muscles are entire and active it is plain that any attempt to form a succedaneum for the acetabulum must be frustrated by continual riding up of the femur (as in Buckminster Brown's case) breaking down or flattening out any new osseous formation. The aim and object of the procedures, which I have carried out and have here described, is to bring the femur to its proper level, and by dividing those muscles which impel it upward to keep it in that place while a new upper lip of an acetabulum, which I make by turning up a semicircle of the periosteum, may have time to form and to become hard.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 92.)

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A CASE

OF

LIGATURE OF THE SECOND PART OF THE LEFT SUBCLAVIAN ARTERY

FOR THE

CURE OF AN AXILLARY ANEURYSM.

BY

RICKMAN JOHN GODLEE, M.B.

Received November 9th, 1891—Read April 12th, 1892.

On July 31st, 1891, I saw, with Dr. J. M. Bruce and Dr. Taylor, of Tooting, a gentleman, aged 66, suffering from a large aneurysm involving the upper part of the left axillary artery. It was evidently thin-walled, and pulsated very forcibly. The tumour projected into the axilla, and, although it was not to be felt above the clavicle, the pulsation in this situation was much more forcible than that on the opposite side; and we therefore concluded that the third part of the subclavian artery was, if not actually involved in the aneurysm, at all events larger than normal. The pain in the arm, though at times considerable, was not excessive, and, indeed, more complaint was made of a certain amount of anæsthesia of the hand.

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Our patient, who is a hearty man for his age, is short, stoutly built, with a short thick neck, and a fair amount of subcutaneous fat. He spent over thirty years of his life at Bathurst on the West Coast of Africa, where, strange to say, he enjoyed good health; in fact, he has never suffered from any serious illness in his life, with the exception of one attack of intermittent fever whilst he was in Africa, in 1856. His occupation did not lead him to perform any great muscular exertion, but, when a young man, he went in for rowing and other forms of athletic sports rather energetically. He never suffered from syphilis or rheumatism.

The first symptom of this illness appears to have been a palpitation in the præcordial region, which, he says, was very violent, and which he first noticed about two years ago, and about the time of its first appearance he, on one occasion, fainted. The palpitation, he is positive, was not in the situation occupied by the aneurysm, but lower down, and in course of time it subsided. The next trouble, which began three months before we saw him, consisted of a numbness in the left arm and hand, which gradually became worse, so that at times he let fall his glove or umbrella; but these symptoms only occurred after he had been walking about for five minutes, and quickly disappeared when he sat down. The pulsation of the aneurysm now became quite apparent, and he assured us that it was much less severe than the palpitation of which he had at first complained.

Dr. Bruce's account of the state of the thorax is as follows:—"Præcordial impulse palpable in the fifth space, just within the left vertical nipple line. No pulsation of any kind in the region of the arch of the aorta. Area of superficial præcordial dulness is of ordinary extent. Over the base of the heart a systolic murmur, and a loud, somewhat thick second sound. The murmur is conducted along the right carotid artery, and it becomes ill-defined towards the mitral area.

"In the left axillary region of the front of the chest are

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discovered the physical signs of aneurysm, as already described, including visible and palpable tumour, excentric pulsation in systole and palpable shock in diastole, both completely controlled (along with obliteration of the radial pulse) by pressure on the subclavian artery; a systolic sound (not murmur); and a loud, thick, diastolic sound. Over the left clavicle, in the situation of the subclavian artery, a whiffing systolic murmur, and the same loud, thick, diastolic sound. The diastolic sound is also audible over the back of the left chest.

"No signs or symptoms of pressure on the trachea, bronchi, œsophagus, or recurrent laryngeal nerve. Radial pulse symmetrical at the two wrists, quiet, regular, somewhat small, very soft, sudden, without thickening of the vessel wall. No signs of venous obstruction."

His general condition was good, though he had for some time been losing weight, three stone in all since the beginning of his illness; but, at his age, we were not disposed to recommend surgical interference without giving Tufnell's treatment a trial; although our patient, who was of a restless disposition, very much resented the idea. He was accordingly put to bed, and placed upon a course of iodide of potassium, and a rigid diet for three weeks,[1] at the end of which time the aneurysm had not apparently diminished in size, and it appeared that while the pulsation was no less below the clavicle, it was somewhat more forcible above this bone. It therefore seemed to me that a further delay would most likely render the application of the ligature quite impossible, so I advised him to submit to the operation, after placing before him, as fairly as I could, the chances of success and failure.

On August 29th chloroform was administered, and I made the incision for ligature of the third part of the subclavian, half an inch above the clavicle, but further in

[1] He began with Pot. Iodid. gr. x, but it was afterwards reduced to gr. v, three times a day. The amount of food was not actually measured, and it was not quite so restricted as was recommended by Tufnell.

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than usual, its inner end reaching halfway or more across the sterno-mastoid. A large vein was divided in the first incision, and then the clavicular head of the muscle was exposed and completely divided across. The artery was then sought, but as it was very much overlaid by the brachial plexus, and as the neck was short and thick, a vertical cut was made, rather more than an inch long, near the inner end of the first incision, which gave much more room, and rendered the exposure of the vessel comparatively easy, after drawing aside with blunt hooks a very large vein which crossed it vertically, and the cords of the brachial plexus. The artery was large and felt very soft, so I divided transversely, with great care, one half or more of the anterior scalenus, whilst anxiously looking out for the thoracic duct and the phrenic nerve, neither of which, however, came into view. The former would probably have been very difficult to see, but I am sure that it was not divided, as no milky fluid welled up in the bottom of the wound. The second part of the artery, thus exposed, appeared to be comparatively healthy. I therefore passed the needle round it from above downwards, threaded with a piece of fine catgut. This was fortunately accomplished without any difficulty. The catgut was then tied to a piece of fine silk, which was attached to a ligature composed of four strands of the finest chromic catgut, which had previously been prepared and tested. The ligature was drawn through and tied, not sufficiently tightly to rupture the internal and middle coats of the vessel. Two pieces of each end were then tied in a reef knot, so as to ensure, if possible, that the original knot should not become loosened, and then all the ends were cut short. The wound, which was now very dry, was washed with a 1 to 500 sublimate solution, and it was all sewn up except about one inch of the outer part. Cyanide of mercury and zinc dressings were applied, and the arm was bandaged to the side, a knitted stocking and some cotton wool being placed over the hand for the sake of warmth.

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Nothing need be said about the healing of the wound, which was complete by the eighth day, except that a certain amount of swelling occurred from extravasation of blood, and that some irritation was produced by the stitches, which were accordingly removed on the fourth day. The discharge of the first twenty-four hours consisted of a considerable amount of bloody serum, but afterwards was practically nil.

The patient kept his bed for a fortnight, and then began gradually to get about again. Whilst in bed he complained of nothing beyond some aching in the upper arm, which still (November 3rd) continues to a slight extent, though it is gradually disappearing. He now looks and feels much better than before the operation, is regaining strength, and can walk moderate distances.

Dr. Bruce and I saw him together on November 6th. He looks rather anxious, and says that he is still weak, and has not regained the flesh which he lost before the operation and during the time he was in bed: he weighs 10 st. 7 lbs., which he thinks is about a stone less than his normal weight. He is, however, cheerful, and has returned to his work at his office in the City. His appetite is excellent and his digestion good. The numbness of his hand has quite disappeared, and his principal trouble is pain in the arm and the front of the chest—no doubt of a neuralgic character, and, it is to be hoped, depending upon the contraction of the aneurysm.

This process of contraction has gone on quite satisfactorily, the induration which remains on the axilla being now quite insignificant. No pulsation is to be felt in any part of the upper extremity, and nothing above the clavicle in the situation of the subclavian, though a feeble pulse, as of the transverse cervical artery, is perceptible a little higher up. Slight thickening still persists above the clavicle; the scar is linear, and the vertical part of it scarcely visible.

So far, then, his condition is quite favorable, but it must be added that there is undoubtedly some faint pulsa-

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tion in the first, second, and third intercostal spaces immediately to the left of the sternum. Dr. Bruce noted at the first examination that none was present; and although it is conceivable that the pulsation, which is now quite easily felt and seen, might have been masked by the much more obvious beating of the aneurysm, it cannot but make us think of the possibility of the gradual development of another aneurysm in the arch of the aorta. In this connection we must also remember the pain in the arm, which he describes as being in the shoulder and elbow especially, partly fixed and partly transitory. The percussion note over the pulsating area is rather higher than that of the corresponding part on the opposite side. The cardiac impulse is diffused, the apex-beat is internal to the nipple, the superficial dulness is less than normal. A murmurish first sound and a thudding second sound are heard at the base, but most loudly over the pulsating area. It is thus pretty certain that he has an atheromatous and dilated aorta, and some atheroma of his aortic valves, but there is nothing definite to point to more serious disease of this vessel. The pupils are normal and equal, and I can detect no inequality in the movement of the two vocal cords; but he has a dry, hard cough (which, however, is not characteristically laryngeal, and which he assures us is just what he has every winter). He also says that his friends tell him that his voice has become weaker, though, for his own part, he does not notice any change in it.

In reporting a case of ligature of the second part of the left subclavian, one is naturally led to try to find out how often it has been done before. I have tried, and am convinced that it would not only be a vain attempt, but that, if it were possible to succeed, it would lead to no useful result. A glance at Poland's excellent article on "Subclavian Aneurysm" in the 'Guy's Hospital Reports' for 1871, vol. xvi, p. 129, yields, indeed, the interesting historical fact that up to that time there had been recorded eight cases of ligature between the scalene

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muscles, of which five recovered. But 1871 was a long while ago—a very long while ago, considering the strides that modern surgery has been making in the interval; while the enormous increase of medical literature makes it almost impossible to anyone, except an absolute bookworm, to gather statistics of the slightest value. I have, therefore, not made the attempt.

Poland concluded, as the result of the careful study of the tables of Norris, Porta, Holmes, Koch, and Le Fort, that the average mortality after ligature of the second and third parts for aneurysm of the subclavian and axillary arteries varied from 40 to 50 per cent.; but it is enough to show how valueless, for us, this conclusion is, when we see a table which he introduced as a matter of course, stating the exact date of the separation of the ligature, and when we find amongst the "consecutive effects" of the operation the most important place assigned to such conditions as these:

Inflammation and suppuration of the tissues in the neighbourhood of the ligature.

Chronic phlebitis.

Pyæmia.

Pleurisy and pneumonia.

For us the questions are (if the justifiability of the operation have been granted)—First, shall we employ an absorbable or a non-absorbable ligature? and second, shall we tie the knot sufficiently tightly to rupture the internal and middle coats of the vessel? and I think that for some years no good will come of collecting statistics, but that we shall do best to simply record our cases, and the reasons which have led us to adopt this or that method of procedure.

I did not rupture the internal and middle coats because I did not feel sure that the artery was healthy enough to bear the strain, and because I thought that, even if it were strong enough, an already dilated vessel would be more likely to become aneurysmal at the seat of ligature if these coats were injured than if they remained

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intact. In doing this I was encouraged by the reports of their experiments on the ligature of arteries by Messrs. Ballance and Edmunds. At the same time I hold the opinion that if an artery be easily accessible, and if its complete exposure do not endanger surrounding important structures, as *e. g.* in the case of the femoral or that of the brachial, the best plan is first to divide the vessel and then to ligature the two ends—a method less likely to give rise to accidents from slipping of the ligature than that of applying first the two ligatures and then cutting between them. But if an artery be not easily accessible I think the safest plan is to apply the ligature tightly enough to rupture the internal and middle coats, if it be healthy; and it is only in the case of diseased vessels that I would recommend the plan adopted here.

I used an absorbable ligature because it seems a more scientific proceeding to leave in the wound a material which will ultimately become completely incorporated with surrounding parts, than one which must remain indefinitely as a possible, if not probable source of mischief; and I used catgut instead of kangaroo tendon, partly because I have no experience of the latter, while I have had plenty to give me confidence in the former and partly because the knot of a piece of kangaroo tendon sufficiently stout for the ligature, seems to me of an inordinate size.

In the performance of the operation easy access to the vessel is obtained by the form of incision I adopted; too much stress cannot be laid on the advantage of having a vertical as well as a horizontal cut. It makes an enormous difference in the accessibility of the deeper parts of the wound. It has been recommended that the phrenic nerve should be sought and held out of the way. If the wound be pretty dry, as in my case, I do not believe that this is advisable, and it really leads one nearer to the edge of the anterior scalenus than it is at all necessary to go. As regards the thoracic duct, it must be remembered that it is in danger as soon as ever the clavicular head of the

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sterno-mastoid has been divided. The best way to avoid it is to cut through the anterior scalenus slightly above the level of the subclavian artery—a plan of proceeding which has other very obvious advantages. I should certainly not join with those who advise that this muscle should be divided upon a director; it seems to me that there is much less likelihood of injuring the pleura or wounding the superior intercostal artery if the fibres of the muscle be picked up one by one in forceps and divided by a knife held horizontally.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 102.)

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ON THE

SYMPTOMS AND PATHOLOGY OF PSILOSIS

(LINGUÆ ET INTESTINI).

BY

GEORGE THIN, M.D.

Received February 6th—Read April 12th, 1892.

For some years past I have been induced to devote attention to the symptoms and peculiarities of a rare climatic disease, examples of which are seen in London in patients who return to their native country in search of health.

One of the symptoms of this disease is a special and characteristic condition of the tongue and mucous membrane of the mouth, and I have had the opportunity of seeing several patients in whom this symptom was a prominent feature. Another characteristic symptom is an irregular and loose condition of the bowels, and to this may be attributed the fact that the class of cases to which I refer has been usually confounded with the various types of disease grouped under the name of *tropical diarrhœa*. The Dutch physicians in Java, where the affection is common, appear to have been the first to call attention to the fact that there is a distinct variety

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of intestinal disease which can be distinguished from the ordinary forms of diarrhœa which are found in the tropics, and to the malady they give the name of "Sprue," a term used in the vernacular in Holland, and in some parts of Scotland, to designate an aphthous condition of the mouth.

Believing that it is desirable to choose a more descriptive name for the affection, and having regard to its most peculiar symptom, I have ventured to suggest that it might, at all events provisionally, be known by medical men as a condition of *Psilosis*.

I have given descriptions of cases of the disease with remarks regarding its treatment and pathology in the 'Practitioner' for September, 1883; and again, more fully, in the 'Practitioner' for 1887 (the latter communications being reproduced in a separate publication); and in a paper in the 'British Medical Journal,' June 14th, 1890.

It is the object of the present paper to record some additional observations that have been made since the date of the last publication to which I have referred. The supplementary information which I am now able to give, although imperfect and fragmentary, is suggestive as regards points of general medical interest, and as the opportunities of carrying on investigations into the nature of the malady are beset with difficulties, there is sufficient reason for bringing the subject somewhat prematurely before the Society.

In the previous publications to which I have referred, evidence is given to show that a peculiar erythematous inflammation of the mucous membrane of the mouth and throat coincides with a diarrhœa of a very peculiar nature, and of an extraordinarily persistent character; whilst in the only case known to me in which a histological investigation was made, parts of the tongue and the whole of the œsophagus were found denuded of epithelium, and the entire mucous membrane of the ileum was found in a shrunken, wasted, and sclerosed condition.

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It is desirable, therefore, that further evidence should be given regarding the condition of the mouth in this affection, and with this object in view two cases are now related in which the characteristic changes were well marked. These cases are selected from others because they form the basis of some further investigations which are described in the paper. In order to illustrate the distinction between this form of disease and what is called ordinary tropical diarrhœa, two cases of the latter affection will be briefly described, and the points of difference shown.

It is also the object of the paper to invite special attention to the colour of the stools in this disease, as the evidence goes to show that colourless or white stools may be passed for a long period, with no affection either of the liver or pancreas. Further, as possibly having connection with this peculiar symptom, some observations on the bacteria present in the stools will be noted.

Mrs. H— consulted me on September 20th, 1890. She informed me that she had been three summers in Shanghai. During the first two summers she had suffered from diarrhœa without any special symptoms, and recovered her usual health with the cold weather. In August, 1889, she was attacked with diarrhœa for the third time, which persisted during the winter and spring, and had never since entirely ceased. This attack had been accompanied from the beginning by an inflamed condition of the mouth and throat, which had persisted equally with the diarrhœa. The motions had been pale from the first, and she had observed that on the few occasions when they were solid these motions were also quite pale. During the voyage to England, on a diet of chicken broth, milk, and biscuits, the symptoms continued with more or less severity, being much aggravated on one occasion after eating chicken.

She had a worn, pale, highly anæmic look; a small, feeble pulse; and in her ordinary clothing only weighed 95 lbs. Apart from the intestinal and mouth symptoms,

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she was free from any sign of visceral disease. She passed several copious pultaceous motions daily, of a greyish-white colour, attended with more or less abdominal uneasiness. Her mouth, and particularly her tongue, were excessively tender and raw, and caused her great distress. An attempt to swallow anything hot—either in flavour or temperature—produced great pain in the mouth and throat. In speaking, the instinctive attempt to prevent movements of the tongue, which were painful, caused the articulation to be thick. The soreness of the mouth was the symptom of which she complained most, and which gave her most distress. Above and to the left of the umbilicus there was a point in the abdomen which at that time, and for many months afterwards, was painful to firm pressure; and during a long period symptoms of abdominal uneasiness of varying intensity continued. A drawing was made of the condition of the tongue at the time, and is now shown to the Society (Plate IV).

She was put to bed, and placed on exclusive milk diet. She was unable to take more than seven glasses, (about three pints) of milk a day. At the end of a week she passed one fairly formed solid stool daily, of a perfectly white colour; and the symptoms in the mouth and throat were greatly relieved. At the end of another week, as the white motions continued solid, and the throat symptoms had entirely disappeared, whilst the mouth was scarcely at all tender, I considered it safe to try an addition to her diet. One raw egg was given in milk on two successive days, and once she had a little water arrowroot. The result was disastrous. The diarrhœa and symptoms of the mouth returned, and it was necessary to put her again on exclusive milk diet, to which she adhered for the next five months, the quantity taken being gradually increased from seven glasses to twelve or thirteen daily, which was her maximum.

During all this period of five months the tongue symptoms continued more or less manifest, but on the whole

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gradually abated. The motions, when either formed or solid—which they usually were—remained quite white. During occasional attacks of slight diarrhœa the fluid that passed was light yellow.

After five months of the milk treatment it was considered safe to give small quantities of beef-tea; and bread was cautiously begun one month afterwards.

During this period there had been occasional slight relapses, mostly due to the influence of cold. One relapse, after she had been able to take beef-tea and a little bread, was distinctly attributable to a small quantity of boiled whiting given in the end of January, and rendered a return to milk and beef-tea necessary for another month. Bread was again resumed cautiously, and in April—ten and a half months after I first saw her—she was able to eat beef, mutton, milk pudding, and chicken; and in another month, exactly one year after I had first seen her, and one year and nine months after the beginning of her attack, she was able to take an ordinary mixed diet.

The pale, colourless, and sometimes greyish motions persisted until the end of December, when, on one occasion, a slightly brownish tinge was noticed. Gradually, as she improved, a slight tinge of light yellow became perceptible in the motions; but it was only after eight months of treatment, when on a diet of milk and beef-tea, that it was noted that the formed motion was of a natural brownish colour. That this return of the natural colour was due to the restoration to health, and not to the diet of milk and beef-tea, is manifest from the fact that on the same diet the motions had been persistently white, or a white occasionally tinged with very light yellow.

During the course of this long illness the symptoms were carefully noted daily by a trained nurse of unusual intelligence and capacity; and the full detailed account of the case appended to the paper is only an abstract of the very much fuller account for which I am indebted to this nurse.

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Soon after the motions became formed, my attention was attracted to a soft, creamy, somewhat consistent mass, which adhered to the upper part of the formed motion. From its appearance and consistence I was at first under the impression that this mass was simply a quantity of undigested cream from the milk which was taken; but an examination made at this time, for and with me by Dr. Wethered, who was kind enough to take an interest in the case, showed that this mass—so far as its solid elements were concerned—consisted apparently of an almost pure cultivation of bacteria.

Dr. Wethered on several occasions made preparations of these bacteria, which were found to consist mostly of a rod-shaped bacterium of what might be described as intermediate size, with a very few large, thick bacteria. The solid part of the motion also contained a large number of bacteria.

This patient for a period of months passed daily a mass of bacteria as the latter part of the stool, often as much as a coffee-cupful, sometimes more, and sometimes less. As the disease abated, this bacterial mass diminished in quantity, and with the disappearance of the disease and the appearance of colour in the motions it entirely ceased.

On ascertaining how largely bacteria entered into the formation of the stool, and how deceptive the ordinary naked-eye appearances had been regarding its composition, I considered it desirable to obtain an analysis of the fæces, which I was enabled to do through the kindness of Mr. Wynter Blyth, to whom I desire to express my thanks. He has contributed largely to whatever value this paper may have by giving freely of his time and special chemical knowledge, by taking a special interest in the peculiar condition of the stools, and by suggesting means by which a study of their constituent elements might be facilitated.

His investigations consisted, first, of an analysis of the milk which the patient took; and secondly, of the fæces.

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Analyses of Milk and Fæces.

(I) *Milk.*

Sample delivered 7 a.m., July 29th, 1890:

	Per cent.
Total milk solids	12·22
„ fat	2·66
„ ash	·60

Sample delivered 5 p.m. (no connection with fæces):

	Per cent.
Total milk solids	12·26
„ fat	3·41
„ ash	·58

(II) *Fæces.*

The fæces had the appearance and consistence of thick Devonshire cream; the total weight was 197·35 grms. The reaction was feebly acid. Absence of albumins. Absence of hæmatin.

On distillation with acetic acid, small quantities of *indol* and *skatol* came over.

(*a*) A small quantity dried at 100° C. showed 82·73 per cent. of water.

(*b*) Ether extracted 7·66 per cent.

(*c*) On addition of a mineral acid to decompose the bile soaps, the residue gave up a second extract of 5·72 per cent. of fatty acids.

(*b*) was saponified up with alcoholic potash, the alcohol got rid of, the soap shaken up with ether, and an extract obtained weighing equal to ·38 per cent. This should be cholesterin, but it presents several distinct differences and requires further investigation. The soap freed from ether and decomposed gave a mixture of cholic, stearic, and palmitic acids; some volatile fatty acids, among which was butyric.

(*c*) was crystallised several times from alcohol; it mainly consisted of cholic acid.

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(*d*) The nitrogen was estimated by Kjeldahl's process. It equalled ·16 per cent.

(*e*) The fæces extracted with hydrochloric acid gave no sign of oxidised sulphur when tested with baric chloride, but the ash contained potassic sulphate, and by fusing with potash and nitre the organic sulphur was found to be equal to ·014 per cent. Most of this is derived from taurin; expressed as taurin, it would be equal to ·057 per cent.

(*f*) The fæces gave a white iron—free ash, total weight equal to 3·27 per cent., ·76 per cent. soluble in water, the latter chiefly potassic and sodic sulphates; the portion insoluble in water was a mixture of calcic carbonate and phosphate.

Hence the whole analysis is as follows:

	Per cent.
Water	82·73
Organic solids	14·00
Ash	3·27
	100·00
Volatile matters:	
Indol	?
Skatol	?
Butyric acids	?
Fixed and neutral fats:	
Palmitin	?
Stearin (cholic acid)	7·28
As soap:	
Cholic acid, with small quantities of other acids	5·72
Body analogous to cholesterin	·38
Taurin	·06
Mineral matters, carbonates, and phosphates of lime	2·51
Sulphates of potash and soda	·76

There are also nitrogenous substances in small quantities. The main peculiarity of the fæces is the absence of the colouring matter (hydrobilirubin) and the large quantity of fatty matters. The whole mass practically consists of bile acids, fats, and ash.

The sulphate is derived from the combustion of organic sulphur matters.

Three pills containing sulphate of iron were given

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at Mr. Wynter Blyth's suggestion, in order to observe whether the ordinary black stools of iron were produced, with a negative result. The only change observed was that the motion was of a yellowish-grey colour, showing the absence of the sulphides usually present in iron stools.

The second case (G. N—) was that of a woman who consulted me in September, 1890. Nine months previously, whilst living in China, she was attacked suddenly by diarrhœa, which had persisted ever since that time. Five months after the diarrhœa began she first became conscious of a sensitiveness of the mouth which had increased to an extreme degree, rendering the swallowing of everything except milk and water exceedingly painful.

When I saw her she was passing five to six greyish or faintly yellow motions of the consistence of gruel, and suffering pain and dryness in the mouth and gullet from the throat to the stomach. The softest boiled grain of rice or sago pained her when swallowed. Apart from the intestinal affection, and the condition of the mouth and throat, she was free from organic disease. The tongue was somewhat swollen and reddened, and presented the appearance shown in the accompanying water-colour drawing (Plate V). There were erythematous patches on the soft palate.

On milk diet the diarrhœa ceased and the throat became well, and after a time a very slight yellowish colour was occasionally observed in the usually perfectly white, colourless, formed stools.

Her history since that time has been a series of periods of improvement after relapses produced by cold, and attempts to take solid food. She has, however, reached a stage in which she can digest bread, fish, and chicken. The motions throughout until January, 1892—fifteen months after I first saw her—have been either white or greyish white, or occasionally very slightly tinged with yellow; but quite recently, simultaneously with a great improvement in all her symptoms, a slightly brownish-yellow colour has been observed in the motions.

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Reduced iron was given after the diarrhœa had been arrested the first time by milk diet, when the motions were solid, but white, in order to test for sulphides. The iron passed quite unaltered in the stools, the ordinary black stools of iron being entirely absent.

After the patient had been three weeks on milk, when the motions consisted of a fairly solid white cylinder, with a slightly softer, creamy or pasty mass following the more solid part of the motion, microscopic examination was made by Dr. Wethered; and it was found that the mass consisted largely of rod-shaped bacteria, very similar in size and general appearance to those described in the case of Mrs. H—.

More detailed accounts of these two cases accompany the paper, but are too long to read before the Society.

It is not necessary to burden this paper with other cases of which I have records, but it may be useful if I abstract from my notes some references to the colour of the motions.

In one case, on a diet of milk, chicken broth, beefsteak, arrowroot, and stale bread, the motions were abundant, semi-fluid, and of a light yellow colour. The same patient at a later period, on a diet of eggs, milk, arrowroot, and beef jelly, had only one motion in two days (the motion being a light yellow); at a still later period, after a partial relapse on a diet of milk, arrowroot, raw eggs, and beef jelly, the motion was divided into two parts as regards colour, one part being slightly yellow, and the other part absolutely white. At another period it was noticed that on a diet of milk, eggs, and occasionally fish, the motions were cream-coloured. It was only after the patient recovered, and was able to digest a mixed diet without relapses, that the motions became a dark or yellowish brown. It was about twenty months from the time I first saw him that he finally recovered. He had been ill two years previously. During the twenty months of which I am cognizant his motions were pale when formed, or very light yellow when they were loose. As the sym-

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ptoms abated they became cream-coloured, and after twenty months they at last became brownish yellow.

In another case, in which the symptoms had lasted indefinitely for some years, and in which recovery had taken place after about six months of treatment, the motions were light, or sometimes bright yellow when they were very loose. The looser they were, the more yellow the colour. When they became solid they were white, and remained white on a digested diet of milk, bread, and fish.

Although now on a diet of coffee, milk, bread and butter, fish, chicken, partridge or pheasant, occasionally mutton and cauliflower, "the motions are still," he writes me, "very pale. They vary slightly, but the usual colour is a yellowish grey, which does not at all approach brown, the first portion of the stool being darker than the latter portion." Although this patient is rapidly recovering, he is still in the stage of occasional relapses, the last of which was probably produced by the mutton; and it is important to note that the condition of incomplete recovery is coincident with the continued absence of the natural yellowish-brown colour of the healthy stool.

In another case of several years' duration, in which the cure has been complete and permanent, a decided colour of the motions did not occur until recovery was so far advanced that the patient had been for two months on a diet of milk, arrowroot, eggs, fish, bread, cauliflower, chicken; and a fortnight on beef once daily.

In another case in which recovery has been complete, and in which for some time the motions were quite white, five months after treatment was begun, and when he was able to take a diet of arrowroot, bread, steak, occasionally boiled mutton, cauliflower with sauce, and a little milk, the hard motions are described as being still very light in colour. In this case the capacity to eat all ordinary foods without relapses coincided with the appearance of dark brown stools.

The absence of the natural yellowish-brown colour in

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the motions, although probably more marked and more persistent in psilosis than in the commoner forms of diarrhœa acquired in the tropics, is also observed in the latter forms of intestinal ailment. In one case of what, for want of a more precise term, may be described as a form of tropical diarrhœa, the solid motion after three weeks of exclusive milk diet was a yellowish white, and after two months of milk diet was a pale yellow; but after only twelve days of beef-tea, bread and butter, and milk, the motion is described as being of a pale brown.

In another case of diarrhœa acquired in the tropics, entirely different from psilosis in its main characteristics, pultaceous, putty-coloured motions were passed. As it was associated with marked yellowness of the conjunctiva, and with a persistent bitter taste in the mouth, it was possibly associated with some disordered action of the liver. When there was much diarrhœa the motions were of a dirty white, and the solid motion passed on milk diet was of a lightish yellow. On a diet of fish, eggs, vegetables, chicken, and fruit, the colour deepened. These two cases are related in detail in the Appendix.

It will be observed that the two cases which have been selected as samples of others yet unpublished or incomplete, entirely confirm the inference drawn from previously published cases to the effect that, in the early stages of the form of intestinal disease known by the Dutch as "sprue," and described by myself as "psilosis," there is a special inflammatory condition of the mucous membrane of the tongue and throat, which is to be distinguished from all other recognised chronic inflammatory conditions of these parts. The appearances are fairly well shown in the two drawings which are submitted to the Society.

This condition of the tongue and mouth may occur in patients who otherwise suffer comparatively little, and in whom the diarrhœa is not a marked symptom; and it is easily distinguishable from the smooth, denuded, beef-steak tongue that may be occasionally observed in the final stages of exhausting intestinal complaints. Not

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only do patients recover from it entirely, but often with remarkable rapidity, whilst its fugitive nature—disappearing sometimes at one part of the mouth, and developing in less than a day on another—gives it a special character.

The nature of the diarrhœa which accompanies psilosis may also, in most cases, be distinguished from the more prevalent forms of ordinary, chronic, tropical diarrhœa. When not acute, its chief characteristic is the copious discharge of a peculiar, dirty greyish, pultaceous mass, with a certain distinguishable, penetrating, disagreeable odour, entirely different from the ordinary fæcal odour. In the acute conditions, when the fluid contents of the bowels are rapidly discharged, the usual colour is a light yellow, in some cases a bright yellow. When the discharges are pultaceous, the food mass having remained longer in the intestine, the yellow colour has disappeared, and is replaced by a greyish, sometimes putty-coloured mass. When the diarrhœa, as a diarrhœa, is arrested, the colour becomes either quite white, greyish white, or cream-white. This absence of colour is more pronounced than occurs in cases of ordinary tropical diarrhœa dependent on other causes. In the latter class of cases there is also a comparative absence of colour. But at a very early stage of recovery a certain amount of yellowness appears in the motions, and increases sooner and with greater intensity than in cases of psilosis; and in cases that do well the normal colour of the motions reappears with much greater rapidity. Two illustrative cases of ordinary tropical diarrhœa, to be used as a contrast to the cases of psilosis, are given in the Appendix to the paper. In one of these the symptoms evidently point to a disordered action of the liver as associated with the prominent symptom of diarrhœa. In both of them, mouth or throat tenderness was entirely absent.

On what does the colourless stools in psilosis depend? The generally accepted opinion that colourless motions are always due to absence of bile has been negatived by

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the important paper contributed to this Society's 'Transactions' by Dr. Walker.[1]

In Dr. Walker's cases the liver was healthy, but the pancreatic juice was entirely wanting, the result being that the patients passed white motions; the inference being, as Dr. Walker has shown, that the brown colour of healthy stools is caused by the action of pancreatic juice on bile. The theory is, as stated by Dr. Walker, that that portion only of the coloured constituents of the bile which has been converted into hydrobilirubin is excreted in the fæces; while the bilirubin, bilifuscin, and biliverdin not so converted are absorbed; and that the colouring matter of the fæces—hydrobilirubin—cannot be produced without the aid of the pancreas.

Now in the cases brought before the Society there was no evidence of any affection of the liver. The skin and conjunctiva were entirely free from jaundice throughout, and the urine was never high-coloured, whilst in the only post-mortem case observed by myself the liver was found healthy. The results of Mr. Wynter Blyth's analysis show that in a typical case the elements of the bile were present in the stools. As regards the pancreas, so far as we know, none of the symptoms from which the patients suffered could be assigned to that organ; and in the post-mortem case referred to, it appeared so healthy to the naked eye that portions of it—perhaps unfortunately—were not put aside for microscopic examination. Further, I have never observed the appearances of free fat in the stools; the pulpy creamy substance, which so simulated the appearance of ordinary cream, but with more consistence, was found, as already described, to consist of an enormous mass of closely agglomerated bacteria, held together undoubtedly by the glia which these organisms form. Mr. Wynter Blyth's analysis shows that a large proportion of the milk-fat consumed was digested. On the 28th of July the total amount of nourishment taken by the patient consisted of about

[1] 'Med.-Chir. Trans.,' vol. lxxii, p. 257.

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70 oz. of milk, or 1984·4 grammes, the mean percentage of milk-fat in this quantity being 60·22 grammes. As the motion passed on the following morning contained only 14·36 grammes of milk-fat, it is evident that 45·86 grammes had been digested during the previous twenty-four hours. This does not indicate any want of pancreatic secretion.

It seems sufficiently clear, therefore, that a patient in whom both the liver and the pancreas are exercising their normal functions may, over a period of months, pass formed colourless motions, although taking a diet compatible with health and strength, and with which a brownish-yellow colour of the stools is usually associated.

Although the facts already given in this paper, and in previous papers published by the author, do not afford a sufficient explanation of this condition, yet taken in conjunction with the results given by Allan Macfadyen, M.D., M. Nencki, M.D., and M. Nieber, M.D., in a remarkable paper entitled "Research into the Chemical Processes in the Small Intestine of Man," they may suggest some theoretical considerations which afford at least a basis of a working hypothesis.

These observers had the rare opportunity of making an extensive series of observations on the food mass which was withdrawn from a fistula of the ileum at the part where it joins the cæcum; and were able also to test the reactions of the mucous membrane, both of the ileum and of the first part of the large intestine; and made an important series of observations on the bacteria which were found to be present in the bowel. They ascertained that in this case the normal reaction of the food mass passing into the cæcum was acid; whilst the mucous coat of the ileum reacted alkaline. The alkaline reaction of the mucosa was increased in the colon, whilst that of the chyme did not begin until the food had passed into the large intestine through the ileo-cæcal valve. The statement found in most text-books that the chyme is already neutralised in the upper part of the small intestine, and

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generally reacts alkaline in the lower part, is apparently incorrect. They further state that a very important and hitherto unregarded function of the intestinal mucosa is the supplying of alkali to the chyme. "An adequate neutralisation of the acid intestinal contents is of essential importance for the normal digestion in the small intestine. Should the mucous membrane furnish too little alkali, a hyperacidity of the intestinal contents must consequently ensue, whereby the separated mucin, instead of becoming mixed with the food mass, is immediately precipitated on the intestinal mucous membrane. In the same manner also the bile acids would be precipitated. Digestion and absorption must thereby suffer."

Now in a fatal case, the particulars of which are given in the 'British Medical Journal' for June 14th, 1890, a thorough microscopic examination was made by Dr. Wethered; and it was found that the mucous membrane of the ileum was sclerosed throughout its whole extent, and was in such a condition that the normal secretion of the membrane must have been absent. If this condition applies to all cases of the disease—and there is much in the clinical history of the cases to show that it is so—we have in this malady an absence of the alkali which is required to neutralise the acid intestinal contents. We shall, therefore, have an abnormal acidity of the chyme during the whole of its passage through the ileum, and when it is discharged into the colon. The observers to whom I have referred, also state that there are certain bacteria in the normal small intestine which decompose the carbo-hydrates, and that the products of the decomposition are ethyl alcohol, the two lactic acids, acetic acid, and succinic acid. An abnormal acidity of the chyme, it may be inferred, will probably interfere with the normal development of these bacteria, and the food which is discharged into the large bowel will, in [illegible] undergone the usual changes which take [illegible] ileum.

[illegible] gues found that the

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alkaline reaction in the food mass begins in the large intestine, and that the alkaline reaction of the mucosa of the colon was more intense than that of the ileum; and each time they examined the fæces of healthy and sick people they found the reaction alkaline.

The fæces submitted to Mr. Wynter Blyth for examination were found to be feebly acid; and in the case of two other patients the fæces were frequently tested, and were found to be invariably acid. That the food mass when it passes into the large bowel is in this disease probably acid, is a matter of inference from the presumed condition of the mucous membrane of the ileum, and from Dr. Macfadyen's observations in his case; but that the fæces when passed were acid is a matter that has been accurately ascertained in three cases.

The mucosa of the large intestine is the habitat of many bacteria, but from what is known of these organisms generally, we must infer that the bacteria of an acid bowel will differ from the bacteria of an alkaline bowel; and it is a matter of fair inference that in the three cases at least in which the reaction of the fæces was tested, the relative numbers of the different bacteria, and possibly also the varieties of these organisms, differed from what is the case in ordinary circumstances. We know that in these cases enormous masses of bacteria were passed in the motions; and in one of the cases to which I have alluded, in which the motions were found to be acid, organisms were present in great numbers, and I succeeded in cultivating several distinct kinds both of rod bacteria and of cocci. We know that when in this disease the motions are driven quickly through the bowel in attacks of diarrhœa, they may be distinctly yellow; and it is when the food mass is driven along with comparative slowness that the colour completely disappears.

It is not an improbable hypothesis that the abnormal products of bacterial growth inevitably associated with an alteration in the reaction of the large intestine, and with great probability with a hyperacidity of the food mass

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in the ileum, are connected with the absence of hydrobilirubin, which is the ordinary colouring matter of the stools. I make no suggestion as to whether the products of bacteria prevent the formation of the colouring matter altogether, or destroy it after it is formed. It is very strictly within the limits of what we know of the laws of bacterial development that the absence of the alkaline secretion of the mucosa of the ileum must exercise an influence on the bacteria present in that part of the gut; whilst an abnormal influence must again be exercised in the large intestine when the fæces are acid.

Whether psilosis, as a disease, may have a bacterial origin must be still a matter of mere speculation; but the clinical facts point to its cause being some specific and special poison, found in some parts of the world and absent in others, which finds its appropriate soil in the mucosa of the ileum. So far as we know, the disease is entirely unknown in Europe, or indeed in any part of the world except certain parts of the East; and it is a plausible assumption that the *causa vera* of the malady is found in certain food or water ingested in these parts. The peculiar and very exceptional effects on the cure or aggravation of the malady, according as the composition of the food mass is affected by diet, may be explained on this hypothesis; foods which are usually considered easiest of digestion often, in this disease, producing disastrous results. The plausible theory of a possible bacterial origin of the disease is certainly not weakened by the discovery of the fact that when the fæces have remained a sufficiently long time in the bowel, there is discharged with the formed motion a large mass of pure bacteria, far in excess of anything that, so far as we yet know, takes place under ordinary conditions.

These are, however, simply suggestions, and I would not wish, by assigning too great a prominence to mere hypotheses, to weaken the import of the facts on which they are based.

Briefly put, the facts are that in an organic but curable

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affection of the mucosa of the small intestine (which in one case has, by post-mortem examination, been proved to be the ileum) the stools remain colourless during long periods of convalescence, although the liver and pancreas are both quite healthy.

The import, therefore, of white or colourless stools is much wider than is usually supposed.

APPENDIX.

THE two cases of Psilosis are related in greater detail than in the previous part of the paper, and for the sake of comparison two cases of a common type of tropical diarrhœa are given.

CASE 1 (Psilosis).—Mrs. H— consulted me on May 20th, 1890. She was a small, pale, anæmic, delicate-looking woman, and informed me that she had been three summers in Shanghai; that she had had diarrhœa each summer, and that this spring she had been sent home for her health. In the summer of 1887 she had morning diarrhœa, which lasted all the summer, and disappeared with the colder weather. In the end of August, 1888, the morning diarrhœa returned. In September she passed a round-worm, after which the diarrhœa ceased. During these two summers she became thinner, but there was nothing wrong with the tongue. Towards the end of August, 1889, the diarrhœa again began, and was worse than in the previous summers. There were about ten motions in the morning, and several during the day, accompanied with abdominal pain. The motions were copious, watery, light in colour, and with a good deal of mucus, the diarrhœa alternating with occasional constipation. She became much thinner. Towards the end of September her mouth became very sore, being raw and painful, and when she swallowed anything hot there was tenderness all down the gullet, and a sensation of great distension after swallowing.

During the winter she was confined of a healthy child, and for a short while after the confinement the diarrhœa ceased, and the tongue got almost well, but as the old symptoms gradually returned she left for England.

Her condition during the voyage varied on a diet of chicken broth,

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milk, and biscuits. The symptoms were much aggravated on one occasion after eating chicken. The soreness of the tongue remained. After being a fortnight in England, at the sea-side, where her condition remained the same, she came to town to consult me.

I found her free from fever, with no symptoms of visceral or thoracic disease, but with a certain amount of tenderness on pressure on the abdomen. The hepatic dulness was normal, or perhaps slightly diminished; the liver was not tender. One particular point, to the left of the umbilicus, was more tender than the rest, and persistently remained so for a very long period. She says that during her illness her motions have always been pale. Several times, for a day or two, since last September she has had solid motions, but they also were pale. Only twice has she observed the colour to be natural during this period. For the last two days she has taken nothing but milk, and for a fortnight previously had taken milk with corn-flour and revalenta. On this diet she had passed one solid motion daily, the colour of which was quite white, but during the whole time the mouth had continued very sore and tender. She remarked that she knew by experience that if, at this time, she had taken bread and butter, in addition to the sore mouth, she would instantly have had a relapse of diarrhœa. She has a tired worn look, and weighs, in her dress and jacket, 95 lbs. She states that her tongue had improved during the last few days considerably. I found it furred in the centre, but red and ragged on the edges, with a somewhat glazed look at parts. There was an herpetic and erythematous eruption on the soft palate. She was put on exclusive milk diet. I have noted that the first motion I saw was copious, pultaceous, of a greyish-white colour, and fermenting, and that she passed several of these the first two days. The tongue continued tender and raw. All attempts to swallow anything hot produced pain in the mouth and throat. The soreness of the tongue caused the articulation to be slightly affected, from an instinctive attempt to prevent its coming in contact with the rest of the mouth. This soreness of the mouth was the symptom of which she complained most, and which gave her most distress.

On the 23rd, after three days of milk treatment, the motion was partly solid, large, and quite white. On the 24th there was a soft white motion, not so copious; and the palate had got well. The patient was not able to take more than seven glasses of milk daily, and felt weak. The milk diet being continued on the 29th, the throat and upper surface of the tongue looked fairly natural in appearance, and the under surface, which until the 28th had continued slightly rose-coloured, had also become almost natural.

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The pulse was small and regular, and about 70, the recumbent position being mostly maintained. She was now able to take eight glasses of milk a day, and was improved in appearance. The one daily motion was formed, solid, large, and of a pale greyish-white colour. The disappearance of inflammation of the mouth made manifest a high degree of anæmia in the mucous membranes. For some days she continued in this satisfactory condition, but the first relapse, whilst under my care, soon took place, and was distinctly due to diet. I had recommended a little arrowroot and a raw egg to be added to the milk, and when she had taken two of these raw eggs the diarrhœa relapsed, and the mouth again became sore. She was put again on pure milk diet, and sent to the sea-side. There she lived on eight glasses of milk a day, spent the morning on the sofa, drove in the afternoon, and walked very little. The mouth did not get well so rapidly this time.

On June 9th she wrote that there was a white spot on the tip of the tongue, another on the inside of the lower lip, and that the sides and back part of the tongue were red, with a somewhat whitish appearance in the centre; and for two days there had been a red patch on the back of the palate. The motions continued to be mostly formed, but still quite white in colour.

June 22nd.—Has been in town for several days. I saw the motions, which were solid, rounded, and formed, somewhat lumpy, very pale in colour; sometimes a very faint tinge of yellow. Since beginning the treatment she has been losing weight at the rate of one pound a week. For the last two days she has had an egg daily in addition to the eight tumblers of milk, and to-day she was ordered a cup of arrowroot and an extra glass of milk. The tongue at this time had a very slight silvery fur, broken on the edges at several places, with almond-shaped patches of a bright rosy red. There were two bright rosy red patches on the under surface of the tongue, one on each side of the raphe, with one small white patch. For several days past the inner surface of the lower lip has been inflamed.

23rd.—After the arrowroot and egg yesterday the motions were not so solid, and there were several in twenty-four hours. Again put on exclusive milk diet.

26th.—Tongue improving. Three motions instead of four on the previous day. They were fluid and white. She takes ten glasses of milk daily.

July 8th.—Since being again on exclusive milk diet the motions have become gradually less loose. Now she passes one formed motion daily as a rule, but some days there are one or two loose

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motions, in one case the colour being described as greyish. She is now taking only seven and a half tumblers of milk a day, and remains in bed. The pulse is 75, small and regular. The tongue, which for some time had been very sore, is now less tender, but is still very red round the edges and at the point. There is also a patch of inflammation on the mucous membrane of the lower lip.

About this time meat suppositories were tried, but were discontinued at the request of the patient, who considered they gave her great discomfort and encouraged diarrhœa. The pain in the stomach at this time was occasionally so severe that starch and opium enemata were given.

12th.—The tenderness in the abdomen is now confined to a spot about the size of a shilling, in the epigastric region, opposite the centre of the false ribs.

This condition continued without much change for some time. The quantity of milk taken varied from eight and a half to ten glasses daily, and the exclusive milk diet was maintained. From the daily record may be taken as samples—

18th.—Tongue feeling decidedly better towards evening. Tenderness in abdomen scarcely to be felt. One motion at 6.30 a.m., partly naturally formed, tinged with yellow; the rest rather pasty-looking, but not quite so white as usual. No pain felt at the time. Ten glasses of milk taken between 4 a.m. and 8 p.m. Feeling better and stronger all day. The tongue continued to vary from day to day.

25th.—States that the tongue is still red on the upper surface at the back, and a new small red patch has appeared on the left side near the tip. There is still a tender spot in the abdomen. Motion to-day was full, partly formed, and the rest a greyish, creamy-looking mass. Took ten and a half glasses of milk.

31st.—Left for the sea-side.

August 2nd.—Tongue much in the same condition, except that on the right side there are three small clefts in the red patch, which is now of a dull red, and appears to be fading, and there is no thick fur in the centre. Tender spot on the abdomen about the same. In the motion at 9 a.m., there were two large hard pieces, slightly yellower than usual. Took eleven glasses of milk.

9th.—Very little redness to be seen on the tongue; the tip quite normal; slight patches at the centre of the back of the tongue and on the left side. Stomach easier. Motion at 11 a.m., full, slightly formed, a tinge yellower than usual. Ten and a half glasses of milk. Feeling stronger and looking better.

16th.—Tongue is not red. Very large formed motion at 9 a.m., of

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a slightly yellow tinge; no early morning pain (the patient often complained of morning abdominal pain). Took ten glasses of milk. During the next week she several times passed a day without any motion. Tongue, which was occasionally slightly tender, is reported on the 23rd as feeling more comfortable, and being pale all over.

No motion on the 24th.

On the 25th there was a motion at 9 a.m., very full, part of it formed (very large), the rest in a heap very slightly formed, and of the usual cream colour. Her diet consisted still of ten glasses of milk a day; and the motions being very large, and occasionally streaked with blood, enemata had to be used.

On the 25th, 26th, and 27th a pill containing sulphate of iron was given, and on the 27th and 28th it is noticed that the "motion is of a yellowish-grey colour."

31st.—Motion, after an enema, preceded by painful straining, and a small quantity of blood was passed on the motion, which was very full, large, and formed; very slightly yellow in colour, and followed by the usual small creamy mass. Ten and a half glasses of milk. For some time motions were only obtained by enemata.

September 7th.—Was able to have a motion without an enema with some straining and pain, there being two or three specks of blood on it. It was all formed and large, but instead of the faint yellow colour was rather greyish. Took thirteen glasses (six pints) of milk.

The daily record during the remaining days of September continued to be much the same. Towards the end of the month she left the sea-side for the country. The motions still consisted of a formed, very pale yellow cylinder, followed by a creamy-looking mass more or less large. She was at this time able to take and digest thirteen glasses of milk a day. There was still some abdominal tenderness.

During the first half of October the colder weather made itself felt injuriously, and the daily motions are reported as being partly formed, but with a large amount of the creamy mass, and on some days some fluid discharge. During the latter half of the month the motions were formed, sometimes hard, occasionally a small creamy mass, the colour remaining the same. Towards the end of the month the colour is reported as being rather yellower. Diet twelve or thirteen glasses of milk daily. There was still some abdominal tenderness.

During November the creamy mass which followed the formed part of the motion, with an occasional liquid discharge, continued intermittingly. On the 13th November the weight was 7 st. 2 lbs.,

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being an increase of 8 lbs. since September 9th. Twice it is noted during the month that a loose motion was of a deep yellow colour. Milk taken during the month, twelve to thirteen glasses daily.

In December she came to town, and was kept in a warm room, and more protected from cold and change of temperature than she had been in the country, with the result that great improvement took place. Beef tea was added to her milk on December 5th, the diet consisting of eleven glasses of milk and two table-spoonfuls of beef tea; and on the 6th twelve glasses of milk and four table-spoonfuls of beef tea. On the 7th arrowroot was given in addition. On that day the dietary consisted of eleven glasses of milk, a coffee-cupful of arrowroot, and three table-spoonfuls of beef tea. For several days after this change of diet there was a formed motion in the morning with a liquid motion in the evening, and for a day or two the arrowroot was discontinued. The dietary on the 10th consisted of ten glasses of milk and four coffee-cupfuls of beef tea. The patient felt stronger. On the 11th it is noted for the first time that the perfectly formed motion passed was of a darker and more natural brownish colour. Bread was then added to the dietary, being gradually increased in quantity; whilst the quantity of milk was diminished, and the quantity of beef tea slightly increased. The dietary from the 11th to the 18th consisted of milk, bread and butter. The motions steadily improved in appearance and consistence. On the 19th a little boiled whiting was added to the dietary, but was withdrawn on the 23rd, on account of a liquid motion. Two days of exclusive milk diet (thirteen and fourteen glasses) cured the diarrhœa; and on the 26th beef tea was again added, and on the 27th bread.

She left town on the 2nd January for the south of England. The dietary consisted from this date until the 22nd January of milk, beef tea, and a small but gradually increasing quantity of bread; the motions being well formed, and very little stomach-ache felt. The tongue was not complained of.

On the 22nd January a little boiled whiting was taken, which was followed by a partly liquid motion, in consequence of which the diet was again restricted to beef tea and milk.

On the 28th January it is noted that the weight was 7 st. 10½ lbs., a gain of 8½ lbs. in the last eleven weeks. The diet was restricted to milk and beef tea until the 27th February, during which period the motions had been formed and of a natural colour and appearance. Bread was again added to the dietary, and from that time her progress was unbroken. There was a good deal of trouble for a time from constipation, for which enemata had frequently to be used.

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The general health rapidly improved. On the 14th March she weighed 8 st., and on the 22nd it was noted that the tenderness in the abdomen, and the stomach-aches, were entirely gone, and that the movements were perfectly natural.

On April 9th she was able to take 4 ounces of beefsteak daily; and on the 19th, it is noted, boiled mutton, milk pudding, and fish. Chicken was added to the diet; and porridge, early in May, to counteract the constipation.

On May 31st it is noted that her dietary included roast mutton, beef, chicken, stewed rhubarb, spinach, butter, eggs, and bread.

I did not see her until midsummer, when I found that the anæmia had entirely disappeared. She looked perfectly well, with a fresh healthy complexion; the only remnant of her illness being an unusual susceptibility to draughts.

On November 17th I had the opportunity of seeing her again, and found that she could eat food of any kind without discomfort. Occasionally she has an uneasy sensation of coldness in the right side, which may last for an hour or two at a time. She associates this with a pain which she states she had in the same part during the whole of her illness.

Case 2.—G. N—, a married woman who had been eleven years in China, consulted me on September 19th, 1890. She informed me that she had excellent health until she returned to the south of China in December, 1889. A fortnight after her arrival she was attacked suddenly by diarrhœa, which continued unchecked by treatment. Every morning about 6 o'clock she had a copious, watery, light yellow motion, accompanied by painful griping. For several days after the attack she had six to eight motions daily, but there was no sickness or other symptom of malaise. Opiates and astringents reduced the motions to one or two daily, but as soon as she left them off the diarrhœa became as severe as at first. Five months after the diarrhœa began she was put on a diet of milk and bananas, with the effect of diminishing the number of motions to two or three soft, white, and frothy stools daily. About the end of May, five months after she was attacked, she noticed that the mouth felt rough, and was very sensitive to sherry and water or anything hot. By the end of June, when living on puddings and chicken broth, her mouth became so bad that she could only swallow with pain and difficulty. Having been put to bed, and placed on exclusive milk diet without good result, she left for England in August. On the voyage home her diet consisted of milk, milk puddings, and cold beef tea, but during all this time the diarrhœa

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and distress in the mouth continued. From the time that the mouth symptoms first began the feeling of soreness and stiffness had been continuous, and so great that she had to get up during the night several times and rinse her mouth with cold water; and her throat was so tender, that from the beginning even rice, unless cooked very softly, gave her pain in swallowing.

When I saw her in London, three days after her arrival, I found she was passing five to six loose motions daily, of a dirty greyish or faint yellow colour, mostly of a gruel-like consistence, each motion being preceded and followed by a considerable amount of pain in the bowels. She had a sensation of pain and dryness in the gullet from the throat to the stomach, and everything she swallowed gave her pain except milk or water. Even rice and sago, although very softly boiled, gave her pain. She got up five to six times every night to rinse her mouth with cold water, to relieve the feeling of dryness and stiffness. She was a pale, anæmic, thin, delicate-looking woman, with a small but regular pulse of 88. There was no fever, or any symptom of organic disease, except the intestinal symptoms; and the urine was normal. Hepatic dulness normal or slightly diminished; liver not tender.

The tongue was free from the usual fur, and marked out by shallow lines into numerous small fields of a pale red colour. On examining it with a magnifying glass the surface, particularly towards the front part, was seen to be composed of a great number of very minute thickly set papillæ, scarcely projecting at all. Solitary rose-coloured papillæ, slightly elevated, with a minute whitish sheath round the root, were dotted over the surface at a distance of one to two millimetres from each other, these papillæ extending over the edge of the tongue towards its under surface. Several dark red erythematous patches were observed on the soft palate.

She was put to bed and placed on milk diet, and after a week there was one formed whitish motion daily; at the same time the symptoms in the mouth became gradually relieved, and within a fortnight of treatment the mouth and throat were comfortable, and the projecting papillæ had ceased to be prominent. On the back part of the tongue, in the centre, there was the beginning of a very slight fur. The tongue was generally less red.

It is not necessary to relate this case in consecutive detail. Its leading features are as follows:

On a diet of five pints of milk daily she suffered from extreme constipation, and she was placed on a diet of milk, arrowroot, corn-flour, biscuits, and bananas, with occasionally bread and butter.

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This diet relieved the constipation, and the motions showed a slight yellowish colour, although the motion was sometimes white without the yellow tint. On the surface of the tongue small islands of thin delicate fur could be observed.

She left London for the country, after three months, in fair health, but received a severe chill on the journey, and all her symptoms relapsed with great severity. During the months of January and February her mouth, especially, became distressingly bad. Eventually, by remaining in bed in a warm room and keeping on exclusive milk diet, these symptoms became relieved, and she again came to town. Since this time her history has been one of fairly steady progress, marked by a series of relapses. Bread, small portions of grilled rump steak, boiled mutton, tapioca, corn-flour, successively produced relapses of diarrhœa with sore mouth, after she had been for a period quite comfortable on milk diet. Three times, including the period previously alluded to, severe prolonged relapses have been produced by chill, each time in travelling.

January 30th, 1892.—Her progress has at last reached a stage in which, with much improved general health, she can, as a rule, take bread, fish, and chicken. Recently a relapse was produced by sucking the juice of an orange on two successive days; but after three days of milk diet the symptoms entirely disappeared.

Early in January she had a slight attack of influenza, and after her recovery, whilst on a diet of bread, milk, and fish, she observed that, for the first time since her illness began, the motions, which were formed, were of a slightly brownish-yellow colour. During the slight relapse which was produced by the orange juice she observed that the soft motion passed was of a yellowish-brown colour.

If we exclude the period preceding the time when she first came under my observation, it is to be noted that for fifteen months the motions, which were often perfectly formed and solid for weeks at a time, were either quite white, or of a greyish-white, or of a very slightly yellowish colour, before the ordinary healthy brownish shade was observed.

In order to test whether the fæces contained sulphides, five grains of reduced iron were given, after she had been a fortnight under my care, and when under exclusive milk diet the motions were formed, solid, and white. The first effect seen was a streak of steel-grey iron on one side of the cylinder-shaped motion, and apparently quite distinct from it. After two days the motions became of a very light slightly greyish tint, as if permeated by minute particles of unchanged iron. After she had taken nine powders, in addition to this greyish tint, partial coatings or sheaths of metallic iron

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adhered to different parts of the surface of the motion. These casts of iron sheathing were two to three inches long, and about half an inch or more broad. The ordinary black colour produced by iron was entirely absent.

As Dr. Begg, of Hankow, has found santonin of benefit in cases of this affection, I tried the drug with this patient, although she told me she had used it without effect before leaving China. She gave it a thorough trial during the long relapse of two months which was produced by exposure to cold in travelling; but it was found to be without the slightest effect in checking the symptoms.

The result of a microscopic examination of the stools has been given in the text.

CASE 3 (Tropical Diarrhœa, June 23rd, 1887).—M. A—, a strongly built man, æt. 54, has been twenty-seven years in Bombay. Has always been a temperate man, and has been home five or six times during that period. Three years ago he had malarial fever, from which as a young man he had not suffered. Whilst in India, in the beginning of May, diarrhœa began, which has lasted ever since. On the homeward voyage he had five or six motions daily, mostly in the morning. He has been home three days, and the diarrhœa continues, there being three to four motions each morning. The dejections are copious, of a very light pale yellow colour, without mucus or blood. There is no pain in the stomach after taking food, and flatulence, to which he was for some time slightly subject, is no longer a symptom. He has an uncertain but not a bad appetite. He lost a stone and a half before leaving Bombay, and has continued to lose weight since. Now weighs about 12 stones or under. Pulse 68, regular; tongue ordinary size, slightly more pink than usual, covered with a whitish fur. Urine free from sugar or albumen, dark in colour, with a deposit of urates. Sleeps well.

There is a diminished area of hepatic dulness. Prescribed one grain of rhubarb with taraxacum twice daily, and every second morning fifteen minims of tincture of rhubarb, and ten grains of Epsom salts. Diet: farinaceous food, milk, eggs, boiled whiting, and beef tea.

June 26th.—Two motions: consistence better. (Yesterday, one in the morning and one in the evening.)

September 26th.—On the whole much improved. For the last ten to fourteen days his bowels have been regular, and the motions natural; but he gets easily chilled, and when he gets chilled the diarrhœa returns, in which case the motions are, as before, pale yellow and watery. To-day his tongue is coated, and he complains

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of a constant bitter taste in his mouth. Prescribed a grain of rhubarb before dinner, and dilute nitro-hydrochloric acid after meals, with a bismuth and soda mixture when there is diarrhœa.

October 4th.—Has a clean tongue, and relishes his food. Still a slightly bitter taste in his mouth.

20th.—Was quite well for a fortnight, but had a slight relapse with the beginning of the cold weather. Is again well, and returns to Bombay immediately. The conjunctiva has a yellowish tinge, and there is still a bitter taste in the mouth.

He had not been long in India when the diarrhœa began gradually to return, and he came home again in the spring. I saw him on May 29th, 1888, and found him looking very ill. He was weak and thin, with a small, soft, and occasionally intermitting pulse. Conjunctivæ were yellow; the tongue natural; hepatic dulness considerably diminished. There was constant diarrhœa to a greater or less extent. The motion which I saw on that day was large, pultaceous, and putty-coloured. Prescribed farinaceous food and milk, and rest.

May 31st.—Motions slightly yellow; conjunctiva less yellow; no fever.

June 4th.—Motions not so thin nor copious, almost white. No fever.

11th.—Twice at night he had taken five grains of sulphur. Diarrhœa much worse, the motions being a dirty white colour, thin, watery, and frothy. To check the excessive diarrhœa six-grain doses of Dover's powder were given, with castor oil. To-day ordered two grains of rhubarb, and one and a half grains of grey powder.

12th.—Same symptoms and treatment.

The symptoms persisting, he was after several days put on pure milk diet, and all medicine was discontinued.

21st.—It is noted that the expression is better. On the tongue, which during the past week had been heavily coated with a fur, the fur to-day is not so thick. There have been two motions, fairly solid but very pale in colour, in twenty-four hours. Had taken sixteen glasses of milk. Pulse 60, fair strength, and regular.

25th.—Continues to look better. Two and sometimes three motions daily, solid but not hard, pale, milk-colour. Takes eighteen to twenty glasses of milk daily.

July 16th.—Pulse 72, strong. Complexion clearer. Face more filled out. Conjunctiva is still somewhat yellow. Thickish white fur on the tongue. Two motions daily.

20th.—For the last four days has had three motions daily, the third one on each day being loose and yellow. The weather has

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been cold and wet, and he has been exerting himself. Tongue still slightly furred. Has gained 4 lbs. in a fortnight, and 10 lbs. during the last five weeks.

With rest, the diarrhœa disappeared. He left London for the country, but had a relapse for five days, during which time he had from four to five motions daily, which had to be checked with opium. The motions had a more yellow colour than when they were solid. Nevertheless he continued to gain weight, having gained three pounds from July 27th to August 8th, when he weighed 11 st. 2 lbs. 2 oz. Continues to take the same quantity of milk.

August 15th.—He wrote that the diarrhœa had entirely disappeared, there being only one motion in twenty-four hours, which was solid, and of a lightish yellow colour. His bowels had not been so regular for many years. Has still, occasionally, a bitter taste in his mouth. This symptom preceded the last relapse already referred to. Takes about five and a half quarts of milk daily.

22nd.—Sensible improvement in the colour of the daily motion. Gained two pounds in the week. Continues same quantity of milk.

September 7th.—Weighs 11 st. 9 lbs. Natural tongue. Hitherto has taken nothing but milk. Allowed arrowroot twice daily, and after four days bread twice daily.

October 18th.—Had a return of diarrhœa which lasted five days, which he attributed to sleeping in badly aired sheets.

31st.—As he was unusually sensitive to changes in the weather, which produced slight temporary relapses of diarrhœa, advised him to go to the Riviera for the winter.

On December 31st he reported that the motions were solid, and with more colour; that he had diminished the quantity of milk, and that he had substituted chicken and milk for porridge, which he had been taking for some time. His diet now consisted of fish, eggs, chicken, with vegetables and fruit, and his weight was 12 st. 7 lbs.

January 21st, 1889.—Reported that in order to keep the daily motion firm he required to retain milk as a considerable element in his diet. From this time he continued to progress and gain weight, and was able to extend his dietary, and within a short period was entirely restored to health.

There had never been at any period soreness of the tongue or throat.

Case 4.—C. H—, æt. 31, consulted me on April 21st, 1890. He had been seven years in the East, and for three years had been in the Straits Settlements. Had not had malarial fever, but had had typhoid fever in Ceylon. In November, 1889, when living up country, he

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was suddenly seized with acute diarrhœa, passing for several days eight or ten motions in the twenty-four hours. The motions were very pale at first, and afterwards some blood was passed. There was some straining and griping; but there was neither fever, nor sickness, nor thirst. On a slop and milk diet the symptoms became alleviated, but up to the present time he has never had fewer than three motions in the twenty-four hours. He has just arrived in England, and latterly has not been dieting, finding that whether he eats slops or solids makes little difference. He has now four or six motions in the twenty-four hours. The discharges are of a gruel-like consistence and a pale yellow colour, and large in quantity. There is a very little straining and griping just at first. He has some discomfort two hours after eating his food; but the tongue has never been sore, nor has there ever been any pain in swallowing. He had lost weight from 10 st. 5 lbs. to 8 st. 5 lbs. but latterly has got slightly heavier, weighing a little over 9 st. Hot and pungent food produces no appreciable pain. The abdomen was soft, not tumid nor tender; liver and other organs normal. Recommended exclusive milk diet.

May 12th.—Been on strict milk diet, taking about four quarts daily. Pulse 85, regular. Tongue fairly clean, being only very slightly furred, the filiform papillæ being distinct. Has had one motion daily, fairly formed, yellowish-white colour, not too copious, and passed without pain. All discomfort has disappeared since he began the milk. Has gained two pounds in the fortnight, and has felt well.

June 2nd.—Motions formed, but not hard, pale yellow colour, not excessive in quantity; one daily, but once or twice a week misses a day. Pulse 80, regular, small. Feels comfortable and stronger. Tongue rather large, showing the marks of the teeth, covered with an ordinary, but not thick, whitish fur; papillæ prominent. Takes from four to four and a half quarts of milk daily. Has gained nine pounds in three weeks. Hepatic dulness continues normal. Ordered breakfast-cupful of arrowroot twice daily for two days; afterwards an addition of corn-flour once daily for two more days. If doing well, to take in addition a cupful of beef tea or chicken broth thickened with corn-flour. Two days afterwards stale bread and butter to be substituted for the arrowroot. In four days two very lightly boiled new-laid eggs to be added. If any supervention of diarrhœa occurred to go back at once to exclusive milk diet.

9th.—Wrote to say that he was going on well. There was a motion once in two days, of a firmer consistence than previously. He now takes beef tea, stale bread and butter, and three quarts of milk daily. Feels stronger, and is getting stouter.

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21st.—Continues to gain weight and strength. Motion is now perfectly formed, pale brown in colour; it is healthy in every respect. Ordered to take three eggs daily instead of two, and to add to his dietary bananas, cauliflower, and boiled or stewed white fish.

On July 9th reports continued health, and that the cauliflower and fish had caused no relapse. Strawberries and boiled mutton are now added to his diet, and they suit him perfectly. When seen on the 9th October he appeared to be in vigorous health. He stated that he occasionally had a sensation after eating as if his food were passing quickly along the bowel, but he had no diarrhœa, and could eat ordinary food without symptoms of dyspepsia.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 107.)

DESCRIPTION OF PLATES IV AND V.

The Symptoms and Pathology of Psilosis (Dr. THIN).

PLATE IV.

The tongue and palate in the case of Mrs. H—.

PLATE V.

The tongue in the case of Mrs. G. N—.

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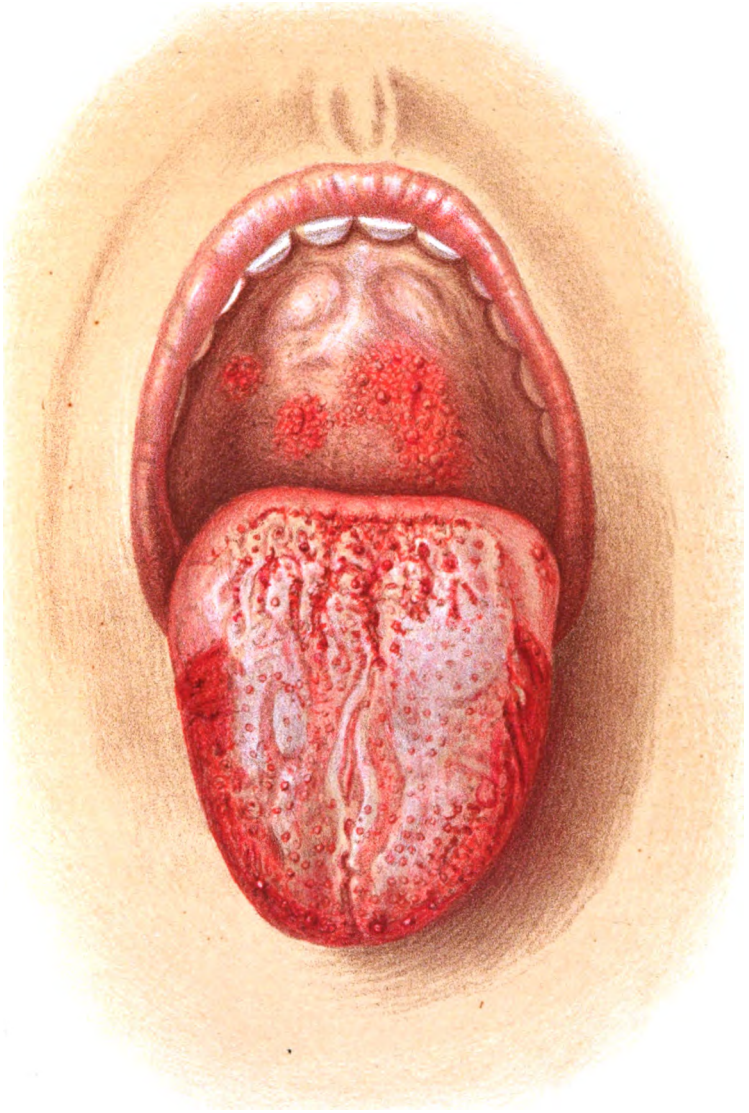

C. P. Burgess del. Mintern Bros. Chromo.

Case of Mrs H.

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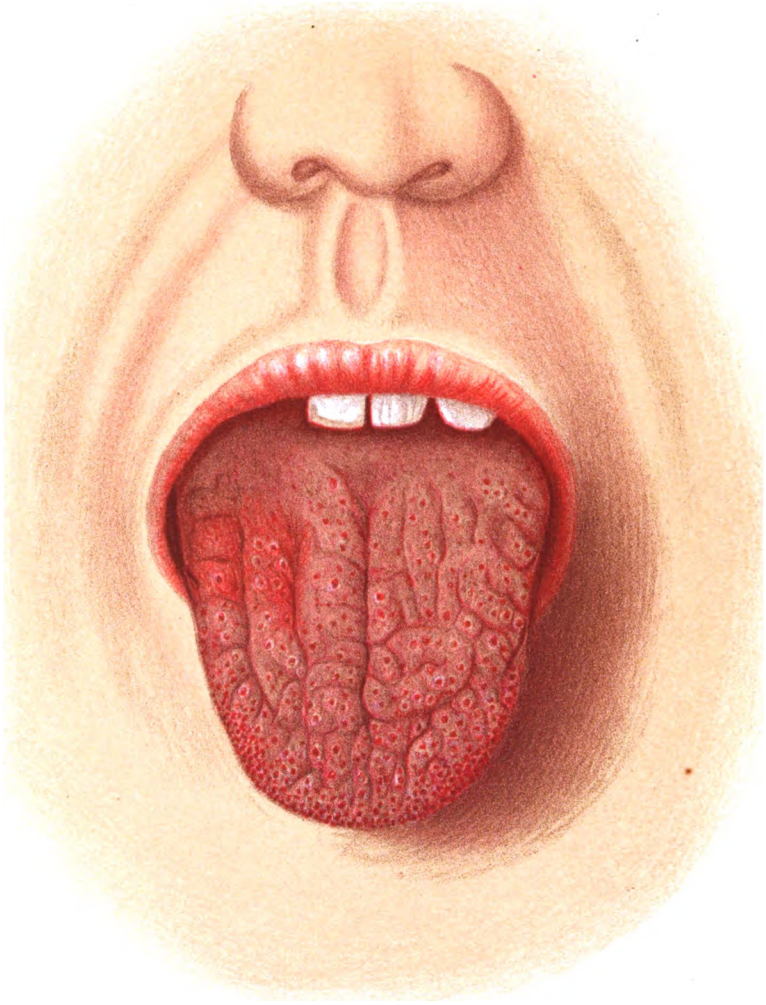

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Case of Mrs G.N.

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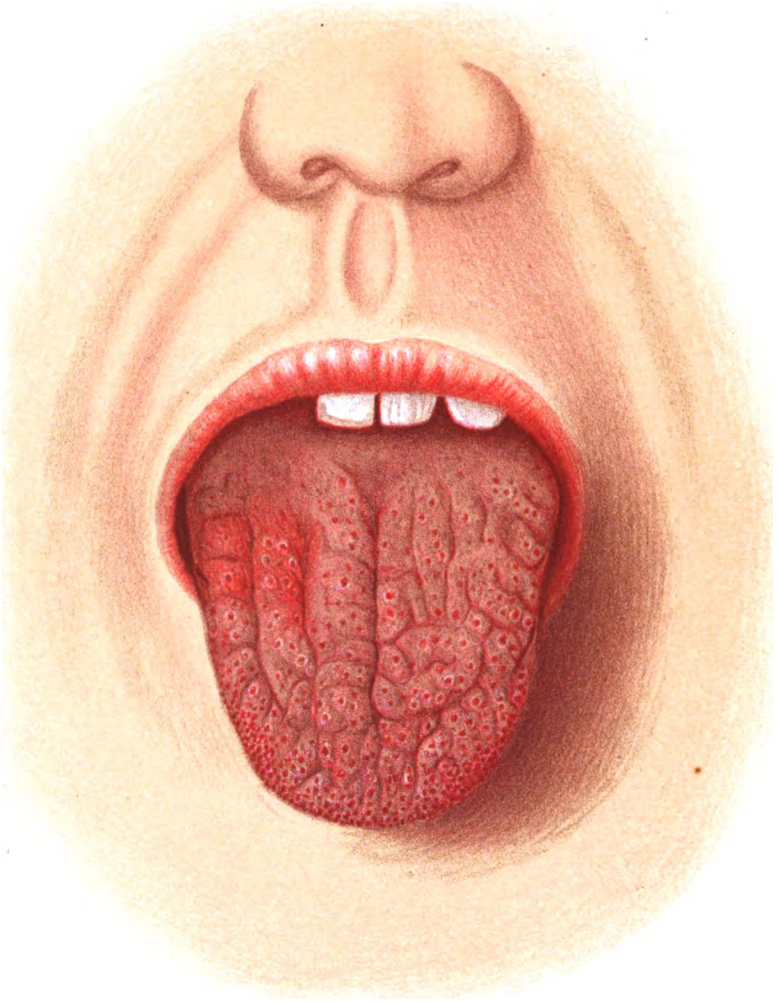

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Case of Mrs G.N.

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ON RENAL DROPSY,

WITH

ESPECIAL REFERENCE TO THE CIRCULATION;

TOGETHER

WITH SOME CONSIDERATIONS RELATING TO PULMONARY APOPLEXY IN RENAL DISEASE.[1]

BY

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Received June 8th, 1891—Read April 26th, 1892.

Since the kidneys provide the chief exit for water, it is to be expected that disease in them such as to obstruct their function should be followed by other disposal of this constituent of the urine. Thus it is a general rule in renal disease, at least in albuminuria, that the dropsy varies inversely with the urine; with the outset of nephritis the diminution of this secretion and the access of dropsy proceed *pari passu*; while should the disease reach an advanced stage, in which the urine is abnormally copious, the dropsy then lessens and disappears. This is

[1] The general conclusions arrived at in this paper were, with the permission of the Council of the Royal Medical and Chirurgical Society, referred to in the Harveian Oration of 1891.

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the rule which generally applies to albuminuria, though even here there are so many exceptional cases that it is obvious that other factors are involved beside the amount of the renal secretion. But if we include other renal and urinary diseases together with albuminuria, we find that conditions apart from the amount of discharge by the kidneys take so large a share in the result that dropsy may present itself when the urine is superabundant, and remain absent though the urine is suppressed. In some of the most intense forms of nephritis, where the urine suffers extreme diminution, dropsy may be nearly or quite absent. Dropsy is commonly absent under obstructive suppression, however complete; while the converse condition is seen with diabetes mellitus, where dropsy often presents itself though the urine is in excess. I have witnessed the development of deep œdema under diabetes with a discharge of fifteen pints a day. With such anomalies awaiting explanation, I will proceed to a brief reconsideration of the leading circumstances of dropsy, more especially when of renal origin.

It is not necessary here to dwell upon the fundamental facts upon which dropsy depends. Dropsy fluid is the exudation from the capillaries which constitutes the lymph, possibly altered by morbid action. This may accumulate either because too much has been poured out by the capillaries, or too little carried off, especially by the veins. So far as dropsy is the result of excess of exudation, and of morbid increase of or addition to the normal lymph, it is to the point to inquire by what processes liquids traverse the walls of living vessels. Of the agencies so concerned there are two which are obvious, and a third which is hypothetical.

1. *Filtration*, or transudation under pressure, acting on all the liquid parts of the blood, colloid as well as crystalloid, the albumen as well as the salts.

2. *Diffusion*, or osmosis, acting on the salts, not on the albumen.

3. *A secretory action on the part of the capillary wall*,

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Pleuræ.									
Sp. gr.	Solids.	Alb.	Mineral		b.	Mineral salts.			Special observations.
			Sol.	Insol.		Sol.	Insol.	Total.	
RENAL DISEASE.									
...	...	...	...	...	56 (est)	·853	·021	·874	Urea ·55 (?).
1008·2	...	·974	·830	·028		...	...	...	Peritoneal F.; urea ·24.
1007·3	1·15	·072 (opalescent)	·695	·021					
1018·5	2·536	1·475	·811	·021					
...	...	...	...	...	5	·866	·025	·891	Œdema, ·30 of urea; uric acid, no trace.
...	...	...	...	...	0	·898	·020	·918	
1010·7	2·050	·742	...	·029	5	·733	·020	·753	
...	...	...	...	...		·769	·025	·794	
1013·2	...	...	·844	·047		·803	·034	·837	
DIABETES.									
...	...	...	...	...	0	·860	...	...	Sugar 1·2 per cent. in œdema; 7·5 in urine at same time.
...	...	...	...	...	5	·870	·023	·893	No trace of sugar in any; urine (P.M.) also free from sugar.
1016·7	4·77	2·105	·80	·045					
...	...	...	...	...	0	·830	·031	·861	Sugar ·21 per cent. in œdema.
EASE OF THE HEART.									
...	...	...	...	...					
..	...	...	...	...		·758	·030	·788	
...	...	...	...	...		·795	·025	·820	
1015·5	4·05	2·909	·766	·029	4	·776	·022	·798	
1012·2	2·841	1·540	·716	·030					
1012·3	3·09	1·910	·776	·035	5	·814	·030	·844	
PULMONARY ORIGIN.									
1012·8	2·986	1·60	·791	·05					
1014·2	3·715	2·230	·780	·032	5	·749	·024	·773	
HEPATIC.									
...	...	...	...	...					
...	...	...	...	...					Ash gave large reaction of iodine.
...	...	...	...	...					
...	...	...	...	...		·774	·03	·804	
LLANEOUS EFFUSIONS.									
1016·8	4·98	4·09	·736	·039					
...	...	...	...	...					

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TABLE II.—*Constituents of Dropsy Fluids in* 100 *Parts* (*Various Observers*).

Authority.	Pericardium.				Pleura.				Peritoneum.				Cellular Tissue.			
	Sp. gr.	Solids.	Alb.	Min. salts.	Sp. gr.	Solids.	Alb.	Min. salts.	Sp. gr.	Solids.	Alb.	Min. salts.	Sp. gr.	Solids.	Alb.	Min. salts.
DISEASE OF THE KIDNEYS.																
Schmidt, 'Lehmann's Chemistry,' ii, 316	...	...	...	...	...	...	2·85	...	...	...	1·13	...	...	...	·36	
Bartels, Ziemssen, xv, 106	1009·6	2·13	...	1·55	...	...	...	...	1009·6	1·56	·25	1·14	1007·6	1·17	Traces	1·06
Schmidt, Gamgee, i, 234	...	...	...	...	...	3·60	...	·75	...	2·10	...	·97	...	1·13	...	·77
Hoppe-Seyler, Gamgee, i, 232	...	...	...	...	...	4·24	2·78	...	...	3·23	1·61	...	...	1·78	·36	·90
Frerichs, 'Diseases of Liver,' ii, 44	...	...	...	...	...	...	...	...	...	2·42	1·10					
Simon, 'Chemistry,' ii, 492	...	...	...	...	...	...	...	...	1007·5	1·93	·81	·80				
„ „ ii, 494	...	...	...	...	...	...	...	...	...	...	...	...	1012	2·4	·70	
„ „ ii, 494 (Heller)	...	...	...	...	...	...	...	...	...	...	...	...	1011	...	Very small	1·01
„ „ ii, 495 „	...	...	...	...	...	...	...	...	...	...	...	...	1010	1·32	Too small to weigh	·88
„ „ ii, 495 „	...	...	...	...	...	...	...	...	...	...	...	...	...	2·48	·54	1·56
DISEASE OF THE HEART.																
Frerichs, 'Diseases of Liver,' ii, 44	...	...	...	...	...	...	...	...	...	1·76	1·18					
DISEASE OF THE LIVER—CIRRHOSIS.																
Lehmann, ii, 316	...	...	1·06	...	...	...	1·85	...	...	...	1·04					
Redenbacher, Day's 'Phys. Chem.,' p. 265	...	...	...	...	...	...	...	...	1018	...	4·20					
„ „ „ „	...	...	...	...	...	...	...	...	1008	...	·84					
Hoppe-Seyler, Gamgee, p. 233:																
1st paracentesis	...	...	...	...	...	...	...	...	...	1·55	·61	·84				
2nd „	...	...	...	...	...	...	...	...	...	1·74	·77	·80				
P.M.	...	...	...	...	...	...	...	...	...	1·66	·61	·81				
Frerichs, ii, 44; average of 6 cases	...	...	...	...	...	...	...	...	...	2·26	1·10					

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such as has been inferred by Dr. Waymouth Reid—a vital rather than a physical process, which may take a part more especially in the operations of disease, but of which as yet we know but little.

It is clear that the products of the first two processes must be different from each other; the result of the third, if it exist, would probably differ from both.

I will now inquire into the composition of dropsy fluids in different diseases, with a view to ascertain whether the effusions present such differences under different circumstances as to suggest that their origin is due to different processes, or such resemblances as to imply that their mode of origin is much the same, whatever the disease.

Such effusions have frequently been examined, as the particulars appended sufficiently show. It has been laid down by Schmidt, and no doubt is generally true, that the quantity of albumen in an exudation is dependent on the system of capillaries through which it occurs; that there is most albumen in pleural effusions, next in peritoneal, least in cellular tissue. By this showing the albuminous character of a dropsical effusion is dependent on its place rather than on its cause. I have ascertained for myself the broad characters, specific gravity, solid matter, albumen, and mineral salts in dropsy fluids from different situations and in different diseases. These observations, though not carried into detail, have the advantage of uniformity of method, and probably afford a fair basis for certain comparisons.

First, as to place: putting aside effusions in the ventricles as not to the present purpose, those in the serous cavities are more albuminous than those in the cellular tissue, the pleural sometimes more so than the peritoneal, sometimes the reverse. Next, as to effusions in the same place under different diseases, the most striking result of the comparison is the smallness of the difference. I will take the amounts of albumen, including in this term whatever is coagulable with heat and acid, and of mineral salts, as ready tests of essential resemblance, and I will include

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in a general average my own results and those of others. First, with regard to albumen: pleural effusions gave under renal disease—not for the present distinguishing between lardaceous and other kinds—1·48 per cent. as the mean of six observations. Pleural effusions under heart disease gave a mean of 2·11 (three observations). As to the peritoneum, renal disease gave 1·09 as the mean of albumen in effusions in this cavity (six observations); heart disease a mean of 2·34 (five observations); liver disease a mean of 1·65 (eleven observations); œdema fluid a mean of albumen of ·34 in renal disease (eleven observations), and a mean of ·35 in heart disease (four observations).[1] Thus in the pleura and peritoneum dropsy effusions of every origin are highly albuminous, though not equally so; the albumen in heart disease being, roughly speaking, about twice as much as in renal disease, that in cirrhosis being intermediate. With regard to œdema fluid, this is but slightly albuminous under any circumstances; as between renal disease and cardiac the difference is not appreciable. As to the fixed or mineral salts, the striking fact about them is the uniformity of their quantity in the effusions of dropsy, whatever their origin and whatever their place. A glance at the annexed tables will show that whenever several fluids have been procured from the same patient, those from the chest, belly, and legs have presented these salts in almost exactly the same amount, and that what preponderance there has been has not been always in the same place. This, however, is less to my present purpose than their uniformity under different diseases. To take the peritoneal fluids as an example, these in renal disease gave ·92 as the average percentage of mineral salts (six observations); in heart disease they gave an average percentage of ·78 (four observations); in cirrhosis an average percentage of ·83 (seven observations). Similarly the fluid of œdema gave under renal disease an average of ·92 (twelve observations); under heart disease an average of ·81 (four observations).

[1] Traces or amounts too small to be weighed are calculated as none.

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The constancy of the mineral salts under different conditions is more strikingly displayed in the tables than in the recapitulation; we cannot fail to infer that whatever be the process of their extrusion, it is much the same whatever the organic cause of the dropsy. The normal percentage of the mineral salts in the *liquor sanguinis* is about ·85, so that dropsy fluids, whatever their place or origin, contain about as much of these crystalloids as the blood from whence they are derived. These salts, chiefly chloride of sodium and the phosphates, transfuse readily, and probably owe their presence in effusions to a simple osmotic action. But it is sufficiently clear that this action will not explain the presence of albumen, which is colloid and little diffusible, and must traverse either as the result of pressure or of some process other than osmosis. Whatever be the process by which albumen transudes, it is in action in dropsy of all kinds, in renal as well as in cardiac and hepatic dropsy. If intra-vascular pressure, as we cannot doubt, is largely concerned in the production of cardiac and hepatic dropsy, the presumption is that it is not unconnected with dropsy of renal origin.

There will be found in the foregoing table, under the heading of Diabetes, a set of estimations which appear to me to have some significance in regard to the origin of dropsy as between osmosis and transudation by pressure, which, although they do not properly relate to renal dropsy, I will venture to refer to as throwing some light upon it. A woman with diabetic coma had within the space of thirty-two hours no less than twenty-two imperial pints of a saline aqueous liquid injected into her veins.[1] Among the results were slight œdema of the legs, and, as ascertained after death, effusions of small amount into several cavities,—18 oz. of straw-coloured serum into the peritoneum, 12 oz. of blood-tinged serum into the pleuræ, 1 oz. of the same into the pericardium. The organs presented general venous engorgement, which in the brain and lungs was intense. I had thought to find the effusions

[1] 'Transactions of the Clinical Society,' vol. xxiii, p. 130.

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especially aqueous, in the expectation that by some process akin to osmosis the blood would be relieved of its superfluous water; but it was clear that increase of pressure within the vessels had more to do with the dropsy than the dilution of their contents. The blood in most of the effusions showed that the pressure had sufficed for corpuscular extrusion; while even the peritoneal liquid, into which no blood had passed, was nearly up to the average in albumen, and not superabundant in water. It is clear that this exudation was brought about less by the alteration in the quality of the blood than by increased intra-vascular pressure.

I will now take into consideration some of the circumstances of renal dropsy with particular reference to the vascular system.

I will place first a class of cases in which total absence or extreme scantiness of urine occurs, with no dropsy or but traces of it. In these there is an apparent deficiency of pressure in the arterial system so far as ordinary observation can testify. There is usually urgent uræmia, and with this much vomiting and inability to take or retain either solids or liquids. A man named Richardson[1] died of calculous suppression which was complete for four days, incomplete for seven. The pulse was at first quiet and regular; latterly it intermitted. He died by syncope. The heart was found to be flabby and uncontracted. He had no dropsy. An exceedingly fat man died after six days of complete suppression of urine due to calculous impaction.[2] He had a little œdema of the legs which did not increase, and was presumably due to old mitral disease. The pulse was always soft, in the earlier course of the illness from 80 to 72; at last it fell somewhat suddenly to 48 without becoming irregular or otherwise different. He then had repeated attacks of syncope, in one of which he died. The left ventricle was found to be entirely uncontracted, which not only de-

[1] 'Renal and Urinary Affections,' vol. iii, p. 951.

[2] Loc. cit., p. 953.

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clared the manner of death, but appeared to show that the fall in the rate of the pulse was not due to digitalis, which was being administered at the time. A lady died of calculous suppression which was complete for four and a half days.[1] The only trace of dropsy was a little puffiness of the eyelids. The pulse on the fourth day numbered 128; it was feeble, jerking, and irregular. She was extremely prostrate, and died, sensible to the last, by cardiac failure. The ventricles were found to be entirely uncontracted. But it is not necessary to multiply cases in proof of what will be accepted as a matter of common experience, that obstructive suppression involves no dropsy or only traces of it, a feeble pulse, and death by syncope.

I will place next another class of cases, in which extreme scantiness or even suppression of urine was dependent on disease of the kidney substance. A boy of nine had intense scarlatinal nephritis, which proved fatal by way of uræmia with convulsions in five days.[2] The urine, which was nearly solid with albumen, averaged almost exactly three ounces a day, or less than a pint for the whole period. Dropsy was almost totally absent—at first none, then only a trace in front of the *tendo Achillis*; no fluid in the serous cavities post mortem. There had been obstinate vomiting of food, medicine, and green slimy matter, what was brought up apparently more in bulk than what was swallowed. The pulse was slow and irregular, the patient under digitalis. A boy of ten passed bloody urine for five days without noticeable diminution; then for sixty-eight hours only half an ounce, still bloody, intensely albuminous, and swarming with large casts and renal epithelium. He then died almost suddenly by way of syncope. There had been somewhat urgent vomiting of food and bilious matter; no diarrhœa. The pulse had been weak and irregular, the temperature low. There was no trace of œdema from

[1] Loc. cit., p. 959.

[2] 'Albuminuria,' edit. 2, p. 95, case of Vallance.

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first to last, nor evidence of fluid in any of the serous cavities. There was no post-mortem, but the existence of intense congestive nephritis was beyond question. A man[1] with albuminuria of lardaceous and syphilitic origin had œdema for a time, but lost it under spontaneous diuresis, vomiting, and diarrhœa. The urine then became so nearly suppressed that the last three days of life gave a total of only two and half drachms. There was no recurrence of the œdema, nor was any dropsical effusion found post mortem excepting a small quantity of serum in the peritoneum. Latterly the vomiting had been persistent, the tongue dry. The pulse became extremely feeble, and fell to 52. The patient remained conscious to the last, and died by asthenia. The heart after death was found to be of small size.

The preceding cases were noted without any special purpose, and, as I now regret, without the use of the sphygmograph; but their testimony is clear, and, if it were necessary, could be corroborated by other cases from medical literature. They show beyond reasonable doubt that with suppression of urine the circulation as a rule is feeble, and dropsy in abeyance. In all there was marked uræmia, in most discharge by vomiting, in some by diarrhœa. They were characterised rather by abnormal excretion than by œdema. We cannot fail to associate the absence of dropsy in circumstances which would appear to invite it with the failure of circulation—in other words, with the diminution of arterial pressure.

To turn now to a condition in which arterial pressure is increased and dropsy present, I come to one which is well known, though perhaps not fully understood. With nephritis, whether from scarlatina or cold, dropsy is usually early present, together with increase of arterial tension, not extreme or conspicuous, but generally decided, less evident to the finger than with the instrument. The late Dr. Mahomed[2] described a pre-albumi-

[1] George Percy.

[2] 'Medico-Chirurgical Transactions,' vol. lvii, 1874.

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nuric stage of nephritis with increased pulse-tension, blood-crystalloids in the urine, and occasionally slight œdema. Dr. Galabin[1] has given tracings in declared but recent nephritis in which, together with albuminuria and dropsy, the tension was decidedly though not extremely increased.

I have been in the habit of late of recording the pulse by means of a Dudgeon's sphygmograph, with a Richardson's lever graduated up to 10 oz.[2] Each observation has consisted of a series of tracings beginning with 1 oz., and going on ounce by ounce until the pressure was enough to stop the pulse, or at least had reached 10 oz. I believe that such a series gives a more definite presentment of the force of the pulse than can be obtained from the mere configuration of one or two lines, however significant. As providing standards of health, I took twenty-one healthy medical students, and fourteen nurses under thirty years of age. The average pulse of the student gives its greatest increment or movement under a pressure of 2·8 oz.; it drops extremely under a pressure of 6·8 oz., and ceases to be perceptible over a pressure of 8·9 oz. The pulse of the nurse gives its greatest movement under a pressure of 3·2 oz., drops extremely under a pressure of 6·4 oz., and ceases to be perceptible over one of 7·5 oz. Thus it may be said in general terms that the pulse of the healthy adult gives its greatest movement at 3 oz., falls greatly at 6½, and stops between 7 and 9.[3]

I annex a table which will show the range of the pulse under nephritis in the acute stage and later, and in the presence and absence of dropsy. Dropsy was present at some time in all the cases referred to excepting two.

[1] 'On the Connection of Bright's Disease with Changes in the Vascular System,' 1873.

[2] Ultimately I found that this was not always enough, and had it weighted up to 20 oz. Only a few observations were made after this change.

[3] These averages are probably high, applying as they do to the time of greatest bodily vigour. Of the twenty-one students, six were over six feet in height. In these and other observations, wherever two or more tracings presented the same degree of movement, the maximum has been taken from the latest.

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TABLE III.—*Pulse in Acute Nephritis in Relation to Dropsy.*

					SPHYGMOGRAPH.		
Name.	Age.	Duration of disease when observation made.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
E. M.	15	10 days	Much œdema	Sounds exaggerated	7	10	Over 10
		14 days	Less œdema	,,	6	10	,,
		26 days	Œdema nearly gone	,,	4	7½	9
		46 days	None	Increase of dulness; second sound accentuated	4	6	7
		2½ months	,,	,,	5	7	7
		5 months	None. Convalescent	Hypertrophy evident	5	8	9
L. M.	18	16 days	Œdema diminishing	Second sound accentuated	5	6	8
		34 days	None	,,	2	—	6
		53 days	Œdema slight but decided	First sound dull; second sound accentuated	4	—	9
		88 days	Much œdema	,,	3	5	7
A. H.	19	17 days	Œdema diminishing	First sound muffled; second accentuated	5	—	8
		24 days	No œdema	,,	3½	—	10
		54 days	Considerable œdema	,,	3	10	Over 10
		91 days	None. Convalescent	,,	3	8	,,
A. W.	37	3 days (?)	No œdema	Normal	—	—	5
		41 days	Slight but decided	,,	4	—	9
		43 days	,,	,,	4	—	9
		47 days	,,	,,	5	—	9
		85 days	Trace. Convalescent	Dulness; second sound exaggerated	4	6	7
G. G.	21	7 days	Œdema	—	1	6	7
		32 days	None	Second sound accentuated	3	6	7
		39 days	,,	First sound prolonged; second accentuated	3	7	8
		62 days	None. Convalescent	Apparently hypertrophied	6	10	Over 10

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Name.	Age.	Duration of disease when observation made.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
M. H.	43	1 day (?)	None	—	7	Over 10	Over 10
		7 days (?)	None. Convalescent	—	5	8	8
L. S.	15	3 months	Much œdema	Dulness; second sound accentuated	5	9	9
A. B.	33	10 days	Much; general	Second sound accentuated	5	9	Over 10
H. S.	18	6 weeks	,,	Dulness	—	—	8
A. G.	26	9 days	Œdema gone under purging	Accentuated	3	4	4
M. R.	25	5 weeks	General	Second sound accentuated	5	8	9
E. S.	41	17 days	Considerable œdema	Sound accentuated	5	—	9
		23 days	Less œdema	Hæmic (?) murmur	6	Over 10	Over 10
		48 days	None	,,	5	—	,,
		70 days	Doubtful trace	Dulness	2	10	,,
H. B.	6	7 days	Slight general œdema	Second sound accentuated	4	Over 10	,,
H. L.	38	2 months	Much œdema fluid in pleuræ and peritoneum	First sound muffled; second accentuated	10	,,	,,
			,,	,,	8	,,	,,
J. B.	20	1 week	None at any time	Pulmonary second sound exaggerated	4	9	,,
		10 days	,,	,,	6	9	,,
		24 days	,,	,,	5	7	9
		36 days	Convalescent	,,	4	Over 10	Over 10
A. M.	29	16 days	General œdema; fluid in pleuræ	Second sound exaggerated; hæmic murmur	4	12	16
		18 days	,,	,,	3	12	16
A. S.	33	6 days	Puffiness not reaching œdema	Pulmonary second sound exaggerated	4	7	10
		11 days	None	,,	5	7	9

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Case of Puerperal Nephritis.[1]

SPHYGMOGRAPH.

Name.	Age.	Date of observation.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
L. F.	20	1 day before labour	Much general œdema; urine highly albuminous	Second sound exaggerated	4	6	18
		4 days after labour	Much less œdema	—	3	6	14
		8 days after labour	—	—	4	6	13
		17 days after labour	No œdema	—	3	7	10
		27 days after labour	No œdema; albumen slight	—	3	7	12
		45 days after labour	—	—	3	7	11
		75 days after labour	No œdema; trace of albumen	Second sound much accentuated	2	7	13
		127 days after labour	Apparently well; slight trace of albumen	Cardiac dulness increased	5	9	14

[1] This case, being of somewhat special nature, is not included in the averages given in the paper.

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TABLE IV.—*Pulse in Chronic Nephritis in Relation to Dropsy.*

SPHYGMOGRAPH.

Name.	Age.	Duration of disease when observation made.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
J. K.	9	4 months	Now none; much formerly	Much hypertrophy	6	—	Over 10
		4½ „	Traces	„	—	—	8
		6 „	Barely a trace	„	3	5	7
		8 „	Œdema increased	„	2	6	9
H. B.	23	7 „	Much general	Dulness, second sound accentuated	7	8	Over 10
M. G.	29	8 „	„	Hypertrophy, final pericarditis	7	Over 10	„
F. A.	3	6 „	„	Hypertrophy	3	8	„
T. H.	25	5 „	None	Dulness, second sound sharp	3	6	7
		+ 1 week	„	„	5	10	Over 10
		6 months	„	„	2	—	„
		7 „	„	„	2	10	„
		7½ „	„	„	4	10	„
		10 „	Trace after walking	„	3	7	9
Miss B.	25	2 years	Much general. Under hard purging	Extremely feeble	—	—	5
M. Y.	49	2 „	Much œdema, ascites, fluid in pleuræ	Natural	6	10	Over 10
J. S.	29	6 months	None	Dulness, second sound accentuated	5	10	10

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Taking first the observations made in acute nephritis and when dropsy was present, it appears that under these circumstances the increase in the force of the pulse was constant and decided though seldom extreme. The sphygmograph gave on an average the greatest movement at 4·7 oz., fell greatly at 8·8, and stopped at 9·4.[1] Thus the pulse of acute renal dropsy conveys to the sphygmograph a force of about an ounce and a half over that of health. And this does not represent the whole increase of blood-pressure, for the elevation lasts longer than in health, as is best displayed with Marey's instrument. Looking at the same series of cases in the absence of dropsy, it would seem that there is less pulse-pressure without dropsy than with it, in the proportion of 8·5 to 9; but this relation is complicated by the beginnings of chronic change, as will presently appear. Proceeding to chronic nephritis, it is to be seen that, whether dropsy be present or absent, the tension of the pulse, or at least the force required to stop it, increases with the duration of the disease. The cases recorded pulsate on an average up to 9·6 oz. Not as yet going beyond nephritis, it is certain that renal dropsy is attended with an increase of arterial pressure, though there may be an increase of arterial pressure and no dropsy. The association of increased tension with nephritis and dropsy is illustrated by the early development of hypertrophy of the heart and arteries—at once a result, a proof, and in some sort a measure of the abnormal state of the circulation. Regarding the hypertrophy as a result of the tension, it is apparent that this is enough to react upon the system and produce pathological change. The hypertrophy is found earliest in the left ventricle, where it often becomes marked within a month of the outset of the renal disease; the arterial change becomes amenable to observation somewhat later, but possibly occurs simultaneously. In Table V, at page 342, are instances of this form of hyper-

[1] In these calculations, where the tracing has not stopped at 10 oz., it has always been reckoned as 11; in some cases it should obviously have been higher.

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trophy produced in from three to ten weeks. Thus we have conclusive evidence that in early nephritis there is enough increase of arterial tension to give rise to structural change, and presumably enough to modify vascular action and transudation.

This increase of tension must necessarily be attributed to difficulty of arterial exit; and next comes the question where precisely the difficulty arises, whether in the arterioles or the capillaries. I am perfectly aware that the arterioles are highly muscular, and that, especially under nervous influences, they have the power of shutting off the blood from their several territories. But here we have to do more immediately with blood than nerve, and it is not unreasonable to suppose that the blood may acquire properties which may interfere directly and immediately with its passage through the capillaries, with which vessels it is brought into more intimate relation than with any others. The capillaries have been shown to be contractile, though they are not muscular; and the fact that œdema presents itself, together with arterial tension, would seem to indicate these vessels as the seat of the impediment. It is not to be doubted that with nephritis the œdema is mainly the result of increased exudation, and that from the capillaries. If the blood-stream were arrested or greatly diminished in the arterioles, we should presume capillary exudation to be diminished rather than increased. It might be suggested as opposed to this presumption that in lardaceous disease œdema appears, though the arterioles are undoubtedly and permanently narrowed. But with regard to this, as will presently appear, the nature of the œdema, as between increased exudation and diminished absorption, is not so clear as to furnish a sure basis for reasoning. Whether in nephritis the capillary hindrance depends on change in the capillaries themselves or in the relation of the blood to them is a question for future investigators; and, indeed, this part of the subject is one in which physicians need further help from physiologists.

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It may be asked whether the series of changes so far indicated is the sole cause of acute renal dropsy. As yet only increased exudation has been had regard to, but there are conditions which at least suggest the possibility of diminished absorption. The frequent association of inflammation of the respiratory organs with acute nephritis cannot but introduce into the problem the question of venous obstruction. I have elsewhere[1] shown what I think is generally recognised, that pneumonia, bronchitis, and pleurisy are, especially in childhood, frequent concomitants of acute nephritis and frequent causes of death. I have more recently had experience of fourteen post-mortems, ten of children, four of adults, in acute renal dropsy of from one week to three months' standing. The condition of the respiratory organs was as follows:

State of organs of respiration in fourteen cases of acute renal dropsy (post mortem).

Natural	1
Pneumonia or pleuro-pneumonia	3
Bronchitis (only)	1
Œdema of lung, congestion, and bronchitis	2
Fluid in pleuræ	1
Fluid in pleuræ with œdema of lung and congestion	5
Fluid in pleuræ with bronchitis	1
	14

It will be seen that the respiratory organs were natural in only one instance, and that in twelve of the fourteen there was bronchitis, congestion or œdema of the lung, or pneumonia.

Now to appeal to clinical apart from post-mortem experience, in sixty-one cases of acute renal dropsy collected during my experience at St. George's Hospital, the Hospital for Sick Children, and elsewhere, noted at

[1] 'Albuminuria,' 2nd edit., p. 45.

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dates varying from four days to three months of the outset, the evidence with regard to the respiratory organs was as follows :

Clinical observations on state of organs of respiration in sixty-one cases of acute renal dropsy, of which thirty-eight were in children under twelve years of age.

No respiratory disturbance noted in . .	18
Cough ; no morbid signs noted in . .	6
Large rhonchus or bronchitis only . .	8
„ „ „ together with dulness	2
Dulness only	7
Evidence of fluid in pleura only . .	4
Fluid in pleura and fine crepitation . .	2
Fine crepitation, coarse or tubular breathing (dulness also noted in five) . .	13
Sounds indistinct	1
	61

It appears that the respiratory organs were abnormal in over two thirds of the number, while in over one third there was evidence of bronchitis or broncho-pneumonia. Thus on every showing it is manifest that inflammatory states of the organs of respiration, particularly as bronchitis or broncho-pneumonia, occur with significant frequency in connection with recent renal dropsy. This cannot but impede the venous return by the lungs, and must enhance the dropsical state by lessening venous absorption. It is occasionally to be observed in such cases that as the lung clears the dropsy diminishes. And it is worth noting in this connection that the exaggeration of the pulmonary second sound in renal disease generally is even more marked than that of the aortic, as if the tension of the pulmonary vessels were affected certainly not less than that of the systemic. But, nevertheless, it is impossible to assign more than a

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secondary place to the respiratory organs in the causation of this form of dropsy, for the dropsy may present itself while these organs are unaffected; and, furthermore, there is usually an absence of superficial venous turgidity, pallor being present rather than congestion. To these considerations must be added the fact that hypertrophy of the right ventricle, which would necessarily result from much and continued pulmonary obstruction, is in nephritis, as in every form of renal disease, little marked compared with that of the left, as if the essential pathology were more systemic than pulmonary. It is probable that the morbid state of the lung and bronchi arises in common with the dropsy as a parallel result of the condition of blood which produces it, rather than that the respiratory lesion is the essential cause of the dropsical.

It is worth remarking that fluid in the pleura stands in a double relation to the dropsy of which it is part, for it necessarily reacts to its increase by way of obstruction by pressure on the venous outlet.

So far it appears that acute renal dropsy has more to do with the state of the arteries than of the veins; and that it is associated with arterial tension, whether this condition be its cause or only connected with the cause. Considering the not infrequent occurrence of high pulse pressure, apart from renal disease, without the production of dropsy, I cannot think that the increase of tension in renal disease suffices alone to account for it, though constantly associated with it. The most feasible supposition appears to be that the morbid state of the blood produces a condition of the capillaries which our present knowledge does not enable us to define, but which both obstructs the blood and facilitates transudation.

Passing now from acute or recent renal dropsy to that of the more chronic conditions—I do not yet touch upon the lardaceous state—another phase is presented. With the continuance of the disease, and its change from acute to chronic, dropsy lessens while arterial tension increases. The dropsy often goes entirely while the tension becomes

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extreme. This appears to be in contradiction to what has gone before, and I must delay for a moment in the attempt to explain the paradox or reconcile the discrepancy. The disappearance of the dropsy with the prolongation of the disease is a natural process, which is too often attributed to treatment. Diuresis often occurs spontaneously, and must share in the *modus operandi*. But that the quantity of urine passed does not alone determine the presence or absence of dropsy is shown by facts to which reference has already been made—the total absence of dropsy under obstructive suppression; its nearly complete absence in some forms of nephritis where the urine is almost suppressed, but not quite; and, conversely, the establishment even of deep œdema under profuse polyuria in diabetes. It is clear that, in the removal of dropsy, other conditions beside diuresis must intervene; if diuresis removes it, the diuresis must occur under certain conditions. Among the conditions which attend the spontaneous mitigation of renal dropsy are great increase of arterial tension and tangible hypertrophy of the heart and arteries. I would venture to assert as a general law that renal dropsy is antagonised by the cardio-vascular change of renal disease.

It is scarcely necessary that I should do more than appeal to the experience of this Society in support of the position that, be the intermediaries what they may, renal dropsy is usually on the wane when cardiac hypertrophy and pulse-tension have largely declared themselves, and even under the same associations is often totally abolished. Nevertheless I will adduce a few instances which it would be easy to multiply.

Case 1.—Mary T—, 21 years of age, a maid of all work, had slight sore throat, followed by swelling of the legs and scantiness of urine. Three weeks later she came into the hospital with much general œdema, which soon increased and became conjoined with ascites and hydrothorax. The swelling of the belly from the dropsy within and without was enough to tear the true

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skin in lines like exaggerations of the *linea albicantes* of pregnancy; similar but smaller fissures presented themselves on the calves. The urine, at first scanty and albuminous nearly to solidity, became gradually more copious, increasing in the course of six weeks from 8 oz. to 132 oz. as the daily quantity while the dropsy lessened with parallel steps. The peritoneum and the right pleura were tapped and the œdema diminished, until ten weeks after admission all dropsy had disappeared, and she was convalescent excepting that the urine still contained albumen, which finally fell to a trace. Mr. Hale, my clinical clerk, made frequent records of the pulse with the sphygmograph, with the result that some over-tension was present from the first, which increased with the diminution of dropsy, and continued to do so after its disappearance. Evidence of hypertrophy of the heart slowly presented itself, while the second sound became markedly accentuated. The patient was very fat, which probably obscured the hypertrophy for a time, but it was sufficiently clear that this had an inverse relation to the dropsy, as also had the pulse tension. When she was last seen, some months after the outset, the heart appeared to be still large; she was then practically well, except for a minute trace of albumen in the urine.

Case 2.—Rosa B— had severe scarlatinal dropsy in St. George's Hospital, at the age of 5. Eight years afterwards she was readmitted with chronic albuminuria, but no dropsy save a little puffiness under the eyes. She then had diuresis and thirst, hypertrophy of the heart, tension of the pulse and albuminuric changes in the retina.

The puffiness about the eyes subsided, and she left the hospital completely devoid of dropsy.

Case 3.—Francis W— had scarlatina at the age of 5, followed by dropsy, which subsided and left him in apparent health, but with a liability to headache and epi-

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staxis. At the age of fourteen, strabismus appeared, a year later he began to lose strength and memory, and he subsequently became slightly deaf on the left side. At the age of twenty he was found to have albuminuric changes in the retinæ. The urine was then albuminous and superabundant, and himself anæmic, emaciated, and entirely without œdema. The heart was obviously hypertrophied, and the pulse one of the hardest I ever felt, difficult to compress, and almost impossible to stop.

He had occasional vomiting, constipation, and wakefulness, and frequent attacks of pain below the umbilicus, especially after food. Symptoms of peritonitis were presently superadded, and were followed by death. It was found, as had been anticipated, that the small bowel was the seat of albuminuric ulceration and perforation. The kidneys were in an advanced state of granular contraction; the heart was increased in weight to 15 oz., the left ventricle and the columnæ carneæ much hypertrophied. Some arteries which were examined, those of the kidney and pia mater, were much thickened.

Case 4.—Bessie B— had scarlatina at the age of 17, followed by swelling of the face, legs, and abdomen. She recovered sufficiently to resume her work as a general servant at a place where she was over-worked and underfed. At the age of twenty-one she began to have diuresis, together with headache, vomiting, and epistaxis. Admitted into St. George's Hospital, she had bleeding at the mouth independently of the epistaxis, profuse menorrhagia, bruise-like marks on the body, hypertrophy of the heart, and a murmur thought to be hæmic. She was in a condition of much exhaustion and anæmia, with a weak and failing pulse. There was no œdema or other dropsy. She sank a few days later, after much dyspnœa. The urine had never been obtainable. The kidneys were found to be granular and atrophied, the left no larger than a horse-chestnut, the right shrunk but to a less extent. The left ventricle was hypertrophied and contracted; the weight

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of the heart was 13 oz. The lungs were congested and œdematous.

I have elsewhere[1] related the case of Emily T—, who died at the age of twelve with a clot in the brain as large as a goose's egg. She had had scarlatina but no sequelæ were recognised, nor had she ever had dropsy. The kidneys were exquisite examples of granular contraction, with profuse interstitial growth. The heart was enormously hypertrophied, especially the left ventricle, and weighed 8½ oz. The valves were healthy. The arteries as examined in the pia mater showed enormous thickening of their walls, both of the muscular and fibrous coats.

There is a common combination of circumstances which I could exemplify numerously, as no doubt could other Fellows of this Society,—chronic albuminuria with the granular kidney, hypertrophy of heart, arterial thickening and tension of pulse, and either no dropsy at any time or only a little, and that in the past.

To give some instances in brief:

Hannah G—, now 40 years of age, single, was first admitted in 1884 with highly albuminous urine, slight œdema in both legs, venous thrombosis in one; no evidence of cardiac hypertrophy, but some hardness of pulse. In 1887 she was readmitted. The œdema was now either totally absent or present only as a trace. The urine was less albuminous, the pulse hard, the left ventricle tangibly enlarged. In March, 1891, she was again admitted, now quite without œdema, though traces occasionally reappeared until the following July, after which there was none. She was much attenuated, the legs shrunk as if dropsy were a *minus* quantity. The urine contained now only a trace of albumen; it was of low specific gravity and alkaline. The chief troubles were nausea and headache, which were treated with success as uræmic. The cardiac dulness was enormously increased, the first sound muffled

[1] 'Albuminuria,' 2nd edit., p. 194; also p. 308.

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and prolonged, the second accentuated. The pulse was tense to the utmost experience of renal disease; it felt like a tendon larger than the radial artery; it could not be completely stopped with the finger, while under the greatest pressure of the sphygmograph, ten ounces, it gave a bold tracing.

George R—, aged 55, a somewhat intemperate marble polisher, began two years before admission to pass much water, and to be occasionally breathless, faint, and giddy. When seen the respiration was laboured, and markedly of the Cheyne-Stokes character. The veins of the neck were turgid. The heart was greatly enlarged, especially to the left, and had the rhythm of the galloping horse, which signifies reduplication. The first sound was muffled, the second sounds accentuated, the pulmonary most so. The pulse was quick and excessively hard, not to be stopped with the finger, while with the sphygmograph a strongly declared tracing was given under the highest attainable pressure, that of ten ounces. The lungs were full of sharp, moist sounds. The urine was pale, contained a trace of albumen, and deposited uric acid.

There was no history of œdema, nor was any evidence of this or of any other form of dropsy to be detected. He had long suffered from insomnia, and shortly after admission became delirious. Measures suitable in uræmia were enforced, and decided though temporary benefit was obtained from a venesection of 14 oz. He died six days after admission. The post-mortem failed to reveal any dropsical effusion. The kidneys, of unequal size, weighed together 9 oz.; they were red, granular, cysted, and fibrotic. The heart weighed 24 oz.; the hypertrophy was chiefly of the left ventricle, though both were hypertrophied and dilated. The auriculo-ventricular openings were dilated, the valves healthy (see outline of heart, Fig. 31).

Both lungs were loaded with frothy fluid; in the

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lower lobe of the left was a mass of pulmonary apoplexy.

The foregoing cases have been selected as typical. The first (Mary T—) shows the gradual removal of renal dropsy under recovery from nephritis coincidently with hypertrophy of the heart and increase of tension. Three others (Rosa B—, Francis W—, and Bessie B—) display as remote sequelæ of scarlatinal nephritis the establishment of the granular kidney with cardio-vascular hypertrophy, and complete or nearly complete removal of the dropsy which had once existed. In all the arterial tension had been such as to cause hæmorrhagic processes. A fifth case exhibits hypertrophy of the heart and arteries and cerebral hæmorrhage in connection with renal fibrosis, to which no dropsy had, as far as was known, ever been annexed. The remaining cases exemplify, as has been indicated, great hypertrophy of the heart, great arterial thickening safely inferred though not exposed to view, extravagant arterial tension, together with absence of dropsy, absolute or nearly so, at least by the time the cardio-vascular change was fully accomplished.

The general and irresistible conclusion is that cardio-vascular hypertrophy, with the arterial tension which is associated with it, have together an inverse relation to renal dropsy. As this state of the circulatory system establishes itself there is frequently, perhaps generally, diuresis; this may be an intermediary *modus operandi*, and presents a point of view in which the inverse relation may be regarded. General increase of tension, involving as it must the renal arteries, tends to cause increase of urine, so that renal disease becomes its own diuretic.

Observations made during life must be supplemented by others made after death. A large degree of cardiac hypertrophy, as is well known, attends the chronic granular kidney; a smaller but yet a decided degree succeeds upon acute nephritis: with the lardaceous

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kidney there is generally none, never much. As a general rule with albuminuria, not lardaceous, the hypertrophy begins with the disease and increases as it continues. In five or six weeks from the outset of nephritis it is usually recognisable. When it has attained a considerable measure the dropsy, as I have demonstrated clinically, usually lessens or disappears.

I annex a list of well-marked cases of renal disease, showing in each condition the amount of dropsy in relation to the state of the heart.

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TABLE V.

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TABLE V.—*State of Heart P.M. in relation to Dropsy.*

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
Males:						
J. S.	32	Large, white; right atrophied from stone	—	Scanty; albuminous; bloody	Slight	Slight
G. M.	31	Cortices swollen, yellowish; surfaces uneven	16 oz.	Scanty, dark, highly albuminous	Much	Much
J. D.	40	Large, white; acute nephritis	22 oz.	Scanty, dark, highly albuminous	Much	Much
J. R.	18	Large, white	25 oz.	Scanty, pale, highly albuminous	Much	Much
H. H.	15	Large, white	14 oz.	Scanty; highly albuminous; high sp. gr.	Considerable	Considerable
R. W.	7	Large, white	8½ oz.	Scanty; highly albuminous; sometimes bloody	Much	Much
J. G.	2	Large, white; acute nephritis	5 oz.	Albuminous	Much	Much
A. W.	2½	Large, white; scarlatinal	4 oz.	Scanty, dark, albuminous	Latterly 0	Latterly 0
J. M.	7	Scarlatinal nephritis	9 oz.	Scanty, smoky, highly albuminous	Much	Much
C. M.	5	Scarlatinal nephritis	5 oz.	Highly albuminous; bloody	Much	Much
W. W.	22	Large, white, smooth	21 oz.	Scanty; bloody; high sp. gr.; highly albuminous	Extreme general	Extreme
J. M.	12	Large, white	13 oz.	At first highly albuminous, latterly less so	General	Much
J. K.	9	Large, white	15½ oz.	Scanty; highly albuminous	Extreme	Extreme
J. K.	48	Right kidney congestive nephritis; left atrophy from obstruction of artery; aneurysm	Right 8 oz.; left 4 oz.	Scanty; highly albuminous	Much	Much

* This mark indicates that the heart is

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ephritis. No Evidence of Mitral Regurgitation.

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	Much	2 years	2 years	15 oz.*	—	Left ventricle hypertrophied; valves natural; slight pericarditis.
Much	Little	Much	10 weeks	10 weeks	12 oz.	—	Pericarditis; valves natural.
Much	Some	Some	8 weeks	10 weeks	14 oz.	—	Left ventricle hypertrophied; valves natural.
Much	Much	Much	2 mos.	3 mos.	14 oz.	—	Slight hypertrophy of left ventricle; valves natural.
1½ pints	0	0	3 mos.	3 mos.	5 oz.	—	Natural.
Some	0	Some?	10 weeks	14 weeks	6¾ oz.	—	Hypertrophy, especially of left ventricle; valves natural.
0	0	Much	3 weeks	3 weeks	3¾ oz.	—	Left ventricle dilated; valves natural.
0	0	0	1 month	Scarlatina 2½ mos.	2½ oz.	—	Some hypertrophy of left ventricle; valves natural.
0	0	Some	16 days	Scarlatina 9 weeks	5¾ oz.	—	Left ventricle hypertrophied.
Much	Some	0	3 weeks	Scarlatina 6 weeks	3½ oz.	—	Valves healthy.
Full of sero-purulent fluid	0	Great	4 mos.	4 mos.	11 oz.	—	Healthy.
0	0	Ascites	13 mos.	13 mos.	6 oz.*	—	Both ventricles hypertrophied, especially left
8 oz.	0	Much	9 mos.	9 mos.	7 oz.*	—	Hypertrophy of left ventricle; slight of right.
0	0	0	3 mos.	3 mos.	28 oz.*	—	Left ventricle much hypertrophied; valves natural

shown in section in the annexed figures.

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Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
Males:						
A. M.	29	Intensely congested; smooth	14½ oz.	Albumen equal to two thirds; scanty	Considerable	Considerable
Females:						
S. W.	30	Large, white; acute nephritis	16 oz.	Very scanty; albuminous	Much	Much
E. L.	33	Large, pale, congested; acute nephritis	16 oz.	Scanty, latterly suppressed; highly albuminous	Much	Much
E. B.	15	Large, white	15 oz.	Scanty, smoky, highly albuminous	Considerable	Considerable
J. I.	2	Acute nephritis; scarlatinal	3¼ oz.	Albumen bloody, not scanty	Much	Much
A. F.	7	Scarlatinal nephritis	5¼ oz.	Scanty, smoky, albuminous	Much	Much
E. A.	6	Large, mottled, smooth	13 oz.	Scanty; highly albuminous	Much	Much
H. L.	10	Large, white, smooth	7½ oz.	Scanty; nearly solid with albumen	Extreme	Much
C. B.	19	Large, white, smooth	19 oz.	Scanty; highly albuminous	Œdema	Much
M. G.	29	Large, white, becoming granulated	10½ oz.	Extremely scanty, pale, albuminous	Much	Much
F. C.	3	Large, smooth; nephritis	6 oz.	Scanty, smoky, highly albuminous	Extreme	Extreme
M. J.	49	Mottled, smooth; advanced nephritis	15 oz.	Scanty; nearly solid with albumen	Much	Much
V. E.[1]	9 mos.	Acute parenchymatous; smooth	5½ oz.	—	0	0

* This mark indicates that the heart is

[1] This case entered also under Pulm

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Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
Some in both	0	0	12 days	15 days	13½ oz.*	—	Valves natural; left ventricle thought to be hypertrophied.
0	0	Much	6 weeks	6 weeks	13 oz.	—	Soft; mitral orifice contracted.
0	0	0	1 month	4 mos.	12 oz.	—	Valves natural; pericarditis.
Some	0	Much	3 mos.	3 mos.	8 oz.	—	Left ventricle hypertrophied; right ventricle dilated; valves natural.
6 oz.	0	0	6 days	Scarlatina 3 weeks	2¼ oz.	—	Slight thickening of mitral edge.
18 oz.	2 oz.	0	4 weeks	Scarlatina 9 weeks	6 oz.	—	Hypertrophied; valves healthy.
Some	Some	Some	2½ years	2½ years	4½ oz.	—	Left ventricle much hypertrophied; valves healthy.
5 oz.	½ oz.	Some	3 mos.	3 mos.	6 oz.	—	Left ventricle hypertrophied; valves healthy.
0	0	0	10 mos.	10 mos.	10 oz.	Systolic murmur latterly	Recent ulcerative endocarditis.
Much	Some	Much	8 mos.	8 mos.	14 oz.*	—	Hypertrophy of both ventricles, especially left; recent pericarditis.
Much	Extreme	Much	9 mos.	9 mos.	2½ oz.*	—	Left ventricle slightly hypertrophied.
Much	0	Much	2 years	2 years	10 oz.*	—	Apparently normal; valves healthy.
0	0	0	—	Diarrhœa 2 weeks	5½ drms.*	Pulmonary apoplexy	Valves healthy; left ventricle thought to be hypertrophied.

shown in section in the annexed figures.
nary Apoplexy.

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State of Heart P.M. in relation to Dropsy. Granular

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
Males:						
D. J.	45	Well-marked granular	12 oz.	Chronic polyuria; latterly scanty; sp. gr. 1028; albumen = ¼	Some	Some
J. W.	20	Granular scarlatinal	—	Pale; sp. gr. 1011; albuminous; much polyuria	0	0
J. B.	28	Highly granular	14 oz.	Pale; albuminous; latterly nearly suppressed	Slight	Slight
R. H.	55	Granular	12 oz.	Highly albuminous; somewhat scanty; albumen = ¼	Much	Much
G. L.	59	Granular; gouty	6 oz.	Latterly pale and scanty	Some	Some
C. D.	14	Well-marked granular	4½ oz.	Excessive; low sp. gr.; albumen slight	0	0
J. S.	52	Granular, cysted, shrunk	—	Excessive, pale, highly albuminous; sp. gr. 1015	None latterly	0
Mr. T.	50	Granular; capsule adherent	—	Pale; sp. gr. 1012; albumen much	Latterly	Latterly
H. T.	39	Well-marked granular	6 oz.	Excessive; sp. gr. 1011; albumen = ¼	0	0
B. H.	60	Granular; tubal nephritis	—	Early excess, latterly scanty; sp. gr. 1017; albumen = ⅝	Much	Much
W. C.	47	Well-marked granular	7 oz.	Pale; sp. gr. 1013; albumen = ⅛	Traces	Traces
J. B.	79	Advanced granular	7 oz.	—	Slight œdema	Slight
H. T.	43	Granular, gouty	9 oz.	Excessive; sp. gr. 1006; albumen = 1/10	None, save in pleuræ	0

* This mark indicates that the heart is

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Kidney. No Evidence of Mitral Regurgitation.

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart	Mitral regurgitation.	Heart, pericardium, infarcts.
40 oz.	0	0	1 month	1 month	21 oz.	—	Left ventricle hypertrophied; valves natural; recent pericarditis.
0	0	0	—	16 years	15 oz.	—	Left ventricle much thickened; valves normal.
Slight	0	0	13 mos.	13 mos.	18 oz.	—	Left ventricle much hypertrophied; valves natural.
Much	0	Much	2 years	2 years	26 oz.	Much bronchitis	Valves good; mitral slightly thickened, aortic beaded; recent pericarditis.
0	0	0	8 weeks	6 weeks	26 oz.	—	Left ventricle much hypertrophied, right ventricle somewhat.
0	0	0	—	—	8 oz.	—	Left ventricle highly hypertrophied; valves healthy.
0	0	0	—	2 years	22 oz.	—	Greatly hypertrophied, chiefly on left side; valves healthy.
1 pint	0	0	3 mos.	15 mos.	—	Loud reduplication of first sound	Enormous hypertrophy of left ventricle; valves natural.
0	0	0	—	2 years	24½ oz.	—	Left ventricle hypertrophied and dilated; valves natural.
Much	0	Much	12 mos.	Some years	25 oz.	—	Left ventricle hypertrophied; valves natural.
0	0	0	—	—	26 oz.*	Apex murmur 8 years ago, none latterly	Both ventricles hypertrophied; valves natural; recent pericarditis.
0	0	0	—	—	16 oz.*	—	Left ventricle hypertrophied; fat on surface; left valves thickened, but competent.
3 pints	0	0	—	Gout 3 years	23 oz.*	—	Both ventricles hypertrophied; valves healthy.

shown in section in the annexed figures.

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Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
Males:						
C. W.	64	Granular, contracted	10 oz.	Albumen = $\frac{1}{6}$	0	0
E. G.	44	Granular, contracted, gouty	8 oz.	Scanty; sp. gr. 1015; albumen = $\frac{1}{4}$	Much	Much; none p.m.
G. W.	58	Granular, gouty	6 oz.	Pale; sp. gr. 1013; albumen = $\frac{1}{8}$	Slight œdema	Slight
P. C.	35	Horseshoe, granular	8 oz.	Amount normal; sp. gr. 1013; albumen = $\frac{1}{8}$	None seen during life	Trace p.m.
Females:						
A. J.	51	Extremely granular	10 oz.	Alkaline; albumen = $\frac{1}{4}$; sp. gr. 1012; pale, clear	Little	Slight
E. T.	12	Granular; highly fibrotic	2½ oz.	—	0	0
B. B.	21	Granular; one atrophied	—	Polyuria 2 or 3 years; pale, latterly darker; sp. gr. 1018; albumen = $\frac{1}{4}$	0	0
L. L.	62	Granular	4 oz.	Pale; sp. gr. 1012; highly albuminous	Some œdema	Some
M. W.	21	Granular, scarlatinal	3 oz.	Pale, copious; much albumen	Some	Traces
E. F.	38	Granular, wasted	8 oz.	Generally scanty; albuminous	Great generally	Much
E. S.	35	Granular, wasted	7 oz.	Highly albuminous; quantity not known	None	None

* This mark indicates that the heart is

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Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	0	—	—	16 oz.*	—	Left ventricle hypertrophied; some fat on surface; valves healthy.
0	0	Much	5 weeks	Gout some years	10 oz.*	—	No marked hypertrophy; valves healthy.
0	0	0	—	—	22 oz.*	—	Left ventricle hypertrophied and dilated; recent pericarditis.
0	0	0	—	6 mos.	18 oz.*	—	Left ventricle hypertrophied; valves natural.
0	0	0	1 year	Over 1 year	26 oz.	—	Both ventricles hypertrophied; valves natural.
0	0	0	—	—	8½ oz.	—	Left ventricle hypertrophied; valves natural.
0	0	0	—	4 years	13 oz.	—	Left ventricle much thickened; valves natural.
0	0	0	3 weeks	3 weeks	13 oz.	—	Left ventricle hypertrophied; mitral valve somewhat contracted, aortic thickened; no murmur.
Much turbid	0	0	1 year	1 year	14 oz.	Murmur at base (hæmic?)	Cavities dilated; valves natural; recent pericarditis.
2 quarts	4 oz.	Distended; tapped often	7 years more or less	7 years	17 oz.	Mitral murmur for last week or two only	Left ventricle greatly hypertrophied; mitral and aortic valves thickened.
None	None	None	—	?	11½ oz.*	—	Left ventricle hypertrophied; valves healthy.

hown in section in the annexed figures.

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Heart P.M. in relation to Dropsy. Granular Kidney and Nephritis Mitral Murmur, apart from in

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
						Pulmonary
Males:						
J. F.	56	Advanced, granular	14 oz.	Albumen = $\frac{1}{10}$; sp. gr. 1020—1025	Much	Much
T. W.	54	Small, red, granular	—	Scanty; highly albuminous	Much	Much
R. M.	70	Congested; surfaces rough	—	Orange; sp. gr. 1030; albumen = $\frac{1}{3}$—$\frac{1}{4}$	—	0
T. S.	24	Granular; left shrunk, contained stones	8 oz.	Copious; sp. gr. 1010; albumen little more latterly	Much	Much
T. G.	62	Granular, cysted; contained infarcts	12 oz.	Albumen = $\frac{1}{5}$—$\frac{1}{6}$; sp. gr. 1016	Much	Much
W. G.	65	Granular, cysted; infarcts	10 oz.	Scanty, high-coloured; sp. gr. 1022; albumen = $\frac{1}{10}$	Considerable	Considerable
H. E.	54	Granular	14 oz.	Very scanty; highly albuminous	Much	Much
G. R.	55	Granular	9 oz.	Formerly superabundant, latterly thought to be scanty; albumen slight	0	0
Females:						
E. R.	36	Granular, cysted	10 oz.	Clear, albuminous; sp. gr. 1020	Much	Much
E. K.	53	Granular	10 oz.	Scanty, lithatic, highly albuminous	Much	Much

* This mark indicates that the heart is

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with Evidence of Mitral Regurgitation, in Pulmonary Apoplexy or dependent disease of the Heart.

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
Apoplexy.							
Some	Slight	—	Over 6 weeks	8 mos.	26 oz.*	Pulmonary apoplexy	Great hypertrophy of left ventricle; mitral opening dilated; valves natural.
4 pints	—	Much	2 mos.	6 mos.	21 oz.	Pulmonary apoplexy	Both ventricles hypertrophied and dilated; no valvular disease.
1 pint bloody	—	Some bloody	—	6 mos.	21 oz.	Pulmonary apoplexy	Flabby; both sides dilated; valves healthy; roughness on pericardium.
—	—	—	—	3 mos.	18 oz.*	Pulmonary apoplexy	Left ventricle much hypertrophied; right ventricle hypertrophied and dilated; valves natural.
Much	—	Some	—	10 weeks	20 oz.	Profuse hæmoptysis	Left ventricle hypertrophied and dilated; valves natural.
Some bloody	—	Some bloody	4 mos.	4 mos.	21 oz.*	Pulmonary apoplexy; mitral murmur	Left ventricle hypertrophied; right ventricle hypertrophied and dilated; valves efficient, somewhat atheromatous.
Much	—	Much	3 weeks	3 years	17 oz.	Threw up a large quantity of blood, apparently from lungs	Left ventricle dilated and soft; aortic valve slightly calcareous.
0	0	0	—	2 years	24 oz.*	Pulmonary apoplexy	Both ventricles dilated and hypertrophied; valves natural.
—	—	Some	3 mos.	24 years?	15 oz.	Pulmonary apoplexy; systolic murmur at apex	Left ventricle hypertrophied; all valves natural.
Much	—	Much	3 weeks	3 years	17 oz.*	Pulmonary apoplexy	Both ventricles dilated; all valves natural.

hown in section in the annexed figures.

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Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
Females:						
V. E.	9 mos.	Large, smooth, mottled[1]	5½ oz.	Not obtained	0	0
						Other Evidence of
Males:						
J. B.	47	Very small, granular; gout	—	Scanty, turbid, highly albuminous	Little	Little; none latterly
T. E.	54	Advanced granular	5 oz.	Clear, pale; sp. gr. 1009; albumen = ¼	Much; probably pericardial	Much
W. S.	45	Finely granular; cortex shrunk	—	Loaded with albumen and lithates	Much	Much
P. H.	42	Advanced granular; gout	7 oz.	Excessive, latterly scanty; albumen = ¼	Much	Much
W. J.	66	Granular	12½ oz.	Copious; low sp. gr.; slightly albuminous	Much	Much
Females:						
E. F.	37	Granular	8 oz.	Pale; highly albuminous; scanty	Much	Much
J. B.	18	Granular, shrunk	—	Diminished; sp. gr. 1012; much albumen	Slight; latterly none	Slight; latterly none
						State of Heart P.M. in relation to
Males:						
W. P.	11	Large, pale; caseous mass in one; caries	12½ oz.	Increased; low sp. gr.; albumen = ¼	Much	Much
F. G.	52	Marked lardaceous; syphilitic	9 oz.	Scanty, albuminous	Much	Slight
C. T.	28	Large, white, syphilitic	24 oz.	Early excess, latterly quantity normal; highly albuminous	Some	Some

* This mark indicates that the heart is

[1] Child rickety and ill-nourished. Died

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Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	0	—	Diarrhœa 2 wks.	5½ drms.	Many pulmonary apoplexies	Left ventricle thought to be somewhat hypertrophied; valves healthy.
Mitral Regurgitation.							
0	0	0	13 mos.	13 mos.	18 oz.	Systolic murmur at apex	Both ventricles hypertrophied and dilated; aortic valve competent; atheromatous.
—	—	Some dark	2 weeks	?	28 oz.	Lungs congested; liver nutmeg	Left ventricle enormously hypertrophied; mitral region pouched; mitral valve stiff; pericarditis; fluid in pericardium.
Much	0	0	1 month	1 month	22 oz.	Murmur; congestion of lungs	Left ventricle hypertrophied; mitral valve thickened, but competent.
0	0	0	8 weeks	Gout 12 years	36 oz.	Doubtful murmur at apex; lungs congested	Cavities large; great hypertrophy; mitral valve thickened; recent pericarditis.
Much	Little	Ascites	2 mos.	10 years	29 oz.*	Systolic murmur at apex	Left ventricle greatly hypertrophied; right ventricle hypertrophied and dilated; valves natural; recent pericarditis.
2 quarts	4 oz.	Much	6 years	6 years	17 oz.	Systolic murmur at apex	Left ventricle greatly hypertrophied; few patches of thickening on mitral and aortic valves.
0	0	0	5 mos.	2 years	16 oz.	Mitral systolic murmur	Left ventricle extremely hypertrophied; right ventricle dilated; mitral valve slightly thickened.
Dropsy. Lardaceous Kidney.							
0	Much	Much	5 mos.	3 years	4½ oz.	—	Normal.
2 pints	Some	17 pints	7 mos.	13 mos.	11 oz.	—	Normal.
0	0	0	Over 4 mos.	—	12 oz.	Edge of mitral thickened	Otherwise normal; no hypertrophy.

shown in section in the annexed figures.
of diarrhœa.

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Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
Males:						
J. P.	43	Pale, waxy, syphilitic	16 oz.	No polyuria; pale; highly albuminous	Trace	Trace
J. G.	42	Early lardaceous; syphilis and phthisis	—	Highly albuminous	Much	Much
G. B.	7	Large, white; stone in one; suppuration	9 oz.	Highly albuminous, pale; sp. gr. 1020	Much	Much
W. P.	40	Smooth, pale, lardaceous; disease general; syphilitic	—	Very scanty; much albumen	Much	Much
G. H.	35	Well-marked lardaceous; syphilitic	13 oz.	Excess; sp. gr. 1009; much albumen	Trace; latterly 0	Trace; latterly 0
J. S.	47	Large, lardaceous; phthisis	—	Diuresis; highly albuminous	0	0
P. F.	34	Large, white; nephritis superadded; syphilitic	—	Much albumen; nephritis superadded latterly	Slight œdema	Slight latterly
W. H.	17	Well-marked lardaceous; phthisis	16½ oz.	Scanty, highly albuminous; sp. gr. 1020	Much	Much
J. W.	36	Pale, cysted, lardaceous	17 oz.	Nearly solid with albumen; pale; sp. gr. 1012	None latterly	0
W. H.	30	Advanced lardaceous (dysentery)	12 oz.	Trace of albumen; sp. gr. 1010	0	0
A. P.	23	Early lardaceous (bronchiectasis)	18 oz.	Nearly solid with albumen	0	0
T. S.	12	Early lardaceous (psoas abscess)	11½ oz.	—	0	0
Females:						
L. B.	20	Advanced lardaceous; suppuration	9 oz.	Alkaline; highly albuminous; diarrhœa	Much	Much
S. F.	6	Not advanced; caries	6 oz.	"Fair quantity"	Some	Some
C. G.	28	Large, white; syphilitic	20 oz.	Excessive, pale; highly albuminous	0	0
L. A.	15	Well-marked lardaceous; caries	6 oz.	Albuminous; quantity not stated	Some	Trace
M. H.	22	Well-marked waxy; suppuration	21 oz.	Lithatic; highly albuminous	Œdema; latterly 0	Some; latterly 0
E. M.	31	Well-marked lardaceous; syphilitic	20 oz.	Copious; low sp. gr.; highly albuminous	Latterly 0	Some; latterly 0
A. H.	36	Highly lardaceous; phthisis; large vomicæ	13 oz.	Scanty; lithatic; highly albuminous	Much œdema	Much

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Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	0	Over 2 mos.	2 years	11 oz.	—	Pale, flabby; valves natural.
0	0	0	2 weeks	1 year	12 oz.	—	Pale; mitral atheromatous, aortic thickened.
0	0	Ascites	1 year	14 mos.	3 oz.	—	Small, firm, lardaceous
0	0	Much	4 mos.	4 mos.	8 oz.	—	Natural.
0	0	0	Trace 3 years ago	3 years	—	—	Natural in size and structure.
0	0	0	—	8 mos.	8 oz.*	—	Healthy.
½ pint	0	0	Recent	Over 3 years	8 oz.*	—	Healthy.
¾ pint	0	1 pint	7 mos.	12 years	6 oz.*	—	Right side hypertrophied and dilated; valves healthy.
0	0	Some	Some 6 mos. ago	6 mos.	12 oz.	—	Pale, soft; patches of atheroma on left valves.
0	0	0	—	7 mos.	13 oz.	—	Natural.
0	0	0	—	2 years	12 oz.*	—	Normal.
0	Slight	0	—	2 years	5 oz.*	—	Valves healthy.
20 oz.	—	—	3 mos.	13 mos.	6 oz.	—	Healthy.
—	—	—	5 mos.	1 year	2 oz. 6 dr.	—	Slight deposits on aortic valve; otherwise natural.
0	0	0	1 year	8 years	8 oz.	—	Left ventricle thought to be hypertrophied.
0	0	Some	1 month	12 years	—	—	Natural.
—	—	—	3 weeks	6 mos.	11 oz.	—	Left ventricle slightly thickened; mitral valve beaded.
0	0	0	13 mos.	4 years	9½ oz.	—	Valves natural.
0	0	0	5 weeks	2 years	7 oz.*	—	Recent vegetations on aortic; otherwise natural.

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The state of the heart is, of course, from post-mortem observation; the condition as to dropsy, to be contemporary, is mainly post-mortem also, though I have included œdema present during the last few weeks, since this form of dropsy is more noticeable during life than afterwards. The cases are mainly from my own notes, and were taken without selection, as they presented themselves. In some the heart is displayed in section.

I have been in the habit for some time past of drawing sections of the heart in various conditions, and venture to bring some of the results before the Society. I began by procuring transverse sections of the ventricular region,[1] which were cut from the heart, mounted in glass, and then traced upon paper; latterly I have been content to expose a section without detachment, trace it upon glass, and thence transfer to paper. I need scarcely add that great care was taken to make the section always at the same place and in the same direction, at right angles to the septum and as high up as practicable without cutting the valves. With so irregular a body as the heart some discrepancies in the fall of the section were probably inevitable, but nevertheless this method would appear not ill suited to demonstrate the thickness of the walls and the size of the cavities (see figures appended).

With these helps I will gather the more prominent facts from the pathological evidence. With simple nephritis there is hypertrophy, though the duration of the disease does not often allow this to become great. With children this was often extremely evident in the exaggeration of the columnæ carneæ, but it is difficult to express in relation to a fixed standard. After the age of eighteen and upwards, leaving out two cases which were complicated by the atrophy of one kidney, the heart of the male averaged just under 13 oz., that of the female just under 12 oz. If Peacock's estimation be accepted,

[1] Twenty-two such sections were made and mounted by Dr. Delépine, seven by Dr. Penrose. I desire to express my great obligations to those gentlemen.

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and 10 oz. and 9 oz. taken as the respective weights of health, the increase with each sex under nephritis is nearly 3 oz. The sections show no general and obvious departure from the condition of health. In one (Maggie Grant) there was distinct dilatation, together with hypertrophy of both ventricles. As hypertrophy of the heart may be regarded as a sure sign of increased arterial tension, the state of the organ confirms the testimony of the sphygmograph in associating tension with nephritis. With both are conjoined scanty urine and abundant dropsy. Thus abundant dropsy is here associated with increased arterial pressure and slight cardiac hypertrophy.

Proceeding now to the granular kidney, this condition in relation to dropsy must be divided into two classes, one where the heart is little altered except by hypertrophy, the other where it has undergone changes which allow of mitral regurgitation. In the first class, that of simple hypertrophy, the heart of the adult male presents an average weight of 20½ oz., that of the adult female one of 15⅔ oz.

In section it is seen that there is enlargement of both ventricles, both by hypertrophy and dilatation. The preponderating change is hypertrophy of the left ventricle, but in some instances there is increase also of the cavity. The changes in the right ventricle are less marked, not always apparent. In some the cavity is enlarged without corresponding attenuation of the walls, so that some degree of hypertrophy must be inferred. The comparatively small degree of change in the right ventricle is apparent on contrasting renal hearts with those of valvular and other diseases. It will be seen that under renal disease the thickening of the left ventricle will bear comparison with that in any other condition; not so that of the right, which, although somewhat enlarged, is conspicuously less so than in some other states.

As regards simple cardiac hypertrophy with the granular kidney, some degree of dropsy was present in two thirds of the cases (sixteen out of twenty-four), more often in

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trifling than considerable amount, so that the effusion is here small while the cardiac hypertrophy is great. As to the urine, this in many cases was excessive, but not constantly so. Roughly it varied inversely with the dropsy, being usually superabundant when dropsy was absent. Taking the cases where there had been such changes in the heart as to give rise to pulmonary apoplexy or a mitral regurgitant murmur, we find that the average weight of the heart of the adult male becomes 23 oz., that of the adult female 16 oz. The sections show (as the symptoms infer) that dilatation has been largely superadded to the hypertrophy. This is manifest in both ventricles, both processes being the more marked in the left. Dropsy now increases. "Much dropsy," instead of presenting itself in a sixth of the cases, is present in two thirds, and together with this scantiness of urine becomes the rule instead of the exception. In addition to the effects of renal disease we have those of valvular inefficiency, and the dropsy which is superadded is as much cardiac as renal. That the hypertrophied heart of renal disease is apt after a time to give way has long been recognised, but I am not aware that pulmonary apoplexy has attracted attention in this relation. I will venture to bring to notice one or two examples of this association in somewhat more detail than was possible in the table. One instance, that of George R—, has been already introduced (page 340 and Fig. 31).

Case 1.—Eliza R—, aged 36, who had been in St. George's Hospital twenty-four years previously with scarlatinal dropsy, was at last admitted with much and increasing œdema, bronchitis, and dyspnœa. The urine was albuminous. The sounds of the heart were abnormal, the first short and sharp, the second markedly accentuated. I myself never detected a murmur, but Dr. Myers, the Medical Registrar, recorded one systolic at the apex. While in the hospital hæmoptysis presented itself, and continued somewhat freely for several days.

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The dropsy increased, so that it became needful to puncture the legs; after much discharge they became erysipelatous, and finally gangrenous with a fatal conclusion.

The kidneys were found to weigh 10 oz.; their capsules were adherent, their surfaces roughened and lobulated. The cortices were somewhat diminished; they were of a pale reddish colour, and contained cysts. The heart weighed 15 oz.; the left ventricle was hypertrophied, and contained several masses of decolourised coagulum; all the valves were natural. The lungs were congested; in the lower lobe of the right were four masses of pulmonary apoplexy, each about the size of a walnut.

Case 2.—John F—, aged 56, had albuminous urine, increasing œdema, much evidence of hypertrophy of the heart, which had a galloping rhythm involving a reduplication, and at times a soft, systolic apex murmur. Upon these ensued rather severe hæmoptysis, Cheyne-Stokes breathing, and ultimately much delirium. During life there had been doubt as to whether the symptoms were primarily renal or primarily cardiac. After death the only uncertainty was whether the case should be regarded as one of granular kidney with its results, or one of general fibrosis in which the kidneys participated. The kidneys were characteristically granular, though not extremely advanced in this state. They each weighed 7 oz.; they were slightly granular on their surfaces, generally cysted and fibrotic. The heart weighed 26 oz.; both ventricles were hypertrophied and dilated, the left most so. The orifices were dilated, especially the mitral and tricuspid; the valves themselves were healthy excepting a little thickening of the aortic, which, however, was not such as to interfere with its action. A section of the heart is represented (Fig. 33).

In the upper lobe of the right lung was a very large mass of pulmonary apoplexy, several inches in diameter; smaller patches of extravasation were scattered through-

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out the left lung. The liver was congested and nutmeggy.[1]

Case 3.—Emma K—, aged 53, came into the hospital after two years of indefinite bad health and a fortnight of dropsy. The urine was scanty, lithatic, and albuminous to one half. The apex of the heart was displaced to the left, the sounds at first free from murmur, though afterwards a low systolic murmur presented itself at the apex. The œdema became deep and general, and, together with much dyspnœa, there was found to be fluid in the right pleura, and 60 oz. were taken away. Before the operation a small quantity of bright blood was coughed up, and more afterwards until the approach of death, ten days later. The kidneys were found to be red and granular, together weighing 10 oz. The heart weighed 17 oz. The left ventricle was much hypertrophied, and dilated with ante-mortem clot entangled in the musculi papillares. The mitral valve had a healthy edge; there was some atheroma near its base of attachment, but not such as to interfere obviously with the action of the valve. The other valves were natural. The right ventricle was dilated and slightly hypertrophied, the right auricle hypertrophied and dilated and contained ante-mortem clot. The heart is shown in sections (Fig. 30). There were many masses of pulmonary apoplexy in both lungs; the largest occupied nearly the whole of the right middle lobe. The lower lobe of this lung was collapsed.

Case 4.—Thomas S—, a potman, aged 24, was admitted into the hospital under Dr. Cavafy, to whom I am indebted for the use of the case. He had formerly been treated by Mr. Rouse for stone in the bladder. For six weeeks before his last admission he had suffered from headache, vomiting, and shortness of breath. The urine was

[1] There were points of interest in this case to which I have not adverted. They have been described by Dr. Delépine, 'Path. Trans.,' vol. xli, 1890, p. 2.

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abundant, of low specific gravity, at first slightly then more albuminous. He had no dropsy, but much bronchitis, and marked albuminuric retinitis with loss of sight. After death, which was preceded by much dyspnœa and delirium, a stone was found in the left kidney, which was atrophied, dilated, and granular. It weighed only 1 oz. The right kidney was also granular; it weighed 7 oz., and contained an old infarct. The heart weighed 18 oz. The left ventricle was much hypertrophied; the mitral valve showed incipient traces of atheroma, but the edge was normal and the valve practically so. The other valves were normal. The right ventricle was dilated and hypertrophied. The heart is shown in section (Fig. 28). The right lung was congested and œdematous, and presented an old infarct in the lateral aspect of the lower lobe over which the pleura was stained. In the lower lobe of the left was a grey pneumonic patch. The cæcum was extensively ulcerated, with blood in the mucous coat; there were also ulcers in the sigmoid flexure and in the duodenum.

In these cases the pulmonary hæmorrhage presents itself as essentially renal; it was not due to disease of the valves, but of the kidneys. Dilatation of renal origin allowed of regurgitation, to which the hypertrophy gave mischievous force. More commonplace evidence of mitral regurgitation with the advanced granular kidney, independently of disease of the mitral valve proper, is to be recognised in the development of a mitral regurgitant murmur in cases where no disease of the valvular structures has been found after death, though the orifices have no doubt been expanded. The tables furnish examples of this. I can also offer the following as a matter of purely clinical observation. With the advanced granular kidney, hypertrophied heart, hard pulse, and renal asthma, it has more than once happened to me to find a loud regurgitant mitral murmur under the asthmatic attacks when in the intervals there was none. The murmur has

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TABLE VI.—*Pulse in Lardaceous Disease in relation to Dropsy.*

Name.	Age.	Duration of symptoms when observation made.	Dropsy.	Heart.	SPHYGMOGRAPH. Greatest movement at — pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
A. H. Phthisis	36	Œdema 2 weeks	Some œdema on tibia	Sounds short and sharp	3	4	5
		1 month	Ditto	—	3	5	7
		6 weeks	Baggy œdema at ankles	—	3	4	6
		7 „	Much, baggy about feet	—	2	4	6
		1 day later	On the increase	Weight 7 ounces	2	3	5
W. H. Phthisis	17	Dropsy 6 months	Much baggy œdema below knees	Right side dilated; weight 6 ounces	2	5	6
L. S. Phthisis	23	Phthisis 6 months	No dropsy	Cardiac dulness increased	R.	9	10
				Pulse somewhat incompressible	L., 1	4	7
A. E. Syphilis	40	Occasional œdema 7 years	Baggy œdema about ankles. Face free	Much dulness	2	6	6
				—	—	6	6
M. S. Syphilis	33	Progressive 3 months	None	Dulness increased	1	5	6
S. G. Disease of hip; amputation	20	Disease of hip 6 years	Slight œdema; 2 months of leg	No obvious change	1	4	7

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been accompanied with palpitation and increased force of the pulse.

To add to the facts of renal dropsy before attempting to form any general conclusions, it is necessary to say a word about lardaceous disease. I regret that my opportunities of obtaining pulse-tracings in this condition have been few, though the post-mortem observations have been more numerous. The tracings show that arterial tension, so far as it has been examined, is generally below par, and this is the case even though œdema is present.

The low pulse-tension accords with the state of the heart as observed after death, which with adult males gives an average of 10¾ oz., with adult females one of 8½ oz. These are so nearly the normal weights that it may be safely concluded that under lardaceous disease there is generally no hypertrophy. Looking at the heart in section, it would seem that this organ in lardaceous disease is weaker, rather than stronger, than in health; the wall of the left ventricle is somewhat thinner than in health, and the cavity of full size, as if under diastole or slightly dilated. Thus the observations of each kind, upon the pulse and upon the heart, present a general agreement, and appear to warrant the conclusion that whatever be the immediate cause of lardaceous dropsy, increase of arterial tension has nothing to do with it. The change special to the disease has the effect of narrowing the arterioles and presumably impeding the current within them, notwithstanding which, as has been seen, neither arterial tension nor cardiac hypertrophy results. If it be thought that these manifestations are in closer relation to the capillary system than to the arterial we may cease to marvel at the discrepancy. But another fact presents itself which requires explanation. Œdema may occur though the arterioles be thus narrowed and the blood-supply to the capillaries correspondingly diminished. If the œdema be due to excess of exudation we have this increase, while the blood-supply on which it depends is diminished.

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This consideration, together with the position of the œdema, which affects the legs rather than the face, may suggest that the dropsy of lardaceous disease has at least some association with lessened absorption and cardiac failure; but further investigation is needed before any theory of lardaceous dropsy can be confidently advanced, further than that it is essentially different from other kinds of renal dropsy. As with all, it has a general inverse relation to the amount of urine. It is to be noted that lardaceous disease is sometimes obviously succeeded by nephritis, and the symptoms modified accordingly; but this is not common enough to interfere with the general statements which have been made.

To place together the conclusions which are led to by the several lines of the inquiry, it will probably be accepted as not admitting of doubt that—

1. Osmosis alone is not sufficient to account for dropsy, whether renal or of other kinds.

2. With the access of the dropsy of nephritis there is increase of arterial pressure. With a further increase of arterial pressure as the disease becomes chronic, and cardio-vascular changes are established, there is diminution of dropsy.

3. Finally, the heart gives way under the pressure to which it is subjected, with mitral regurgitation, pulmonary apoplexy, and increase or recurrence of dropsy.

All but the first are matters of simple observation, and none, I think, admit of question. As to the explanation of dropsy itself, and of the various changes which go with it, I desire to speak with a full consciousness of the difficulty and complication of the subject, and a sense that nothing can be as yet advanced which is more than provisional. With this admission I will revert in somewhat more detail to some of the points which have been touched upon, and will annex such inferences as present themselves.

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Not only are the laws of osmosis or dialysis insufficient to explain the albuminous effusions of renal dropsy, but the similarity of these effusions to those which depend, as in heart disease, upon modifications of intra-vascular pressure suggests that their mode of origin cannot be entirely dissimilar.

The effects of osmosis are probably to be recognised in the presence of urea and sugar in dropsical effusions under certain circumstances; and this process has been thought to be largely concerned in renal dropsy. Dialysable impurities may be presumed to be abundant in uræmic blood, and dialysis might be thought to be mainly concerned in the attendant dropsy, were not the effusions albuminous. So far as the mineral salts may be taken as the measure of dialysis, this would appear to be much the same in all kinds of dropsy. The connection between dropsy and uræmia as a matter of clinical experience is by no means simple; for this state of blood may be extreme, even unto death, while serous effusion is conspicuously absent.

Anæmia and hydræmia are both to be considered in relation to renal dropsy. Anæmia is so often associated with this that it may be supposed to have some necessary connection with it. It is at least clear that both dropsy and anæmia can be traced, directly or indirectly, to the same cause, in impurity of blood. Anæmia unconnected with renal disease does not always give rise to dropsy, however extreme. Dropsy is usually entirely absent with idiopathic or pernicious anæmia, where it should be abundant could impoverishment of blood by itself give rise to it. With chlorotic anæmia œdema presents itself occasionally and to a slight extent. Here the anæmia is generally more marked than with renal disease, the dropsy less. But there *is* œdema sometimes, and here a consideration of some interest presents itself, though I desire to advance it with caution, as based as yet upon few observations. I found that with chlorotic anæmia, notwithstanding the prevailing debility, the tension of the

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pulse was increased. This observation took me by surprise; but so far as the evidence of eight cases and of Dudgeon's sphygmograph can be trusted, it appeared to be decided. Anæmia was marked in all, œdema present in five. At all stages there was evidence of more than normal arterial pressure, but it is enough to say that movement was obtained on an average up to a pressure of 9·6 oz., 2 oz. beyond the average of the nurse. In one of these cases of chlorotic œdema I may mention that the Medical Registrar discovered a patch of retinal hæmorrhage as another indication of undue arterial pressure. If further and more varied observations should accord with these, we may discern in chlorotic anæmia and the dropsy attending it something beyond mere poverty of blood, and find an analogy with renal disease. In both we have a toxic element, in one from deficiency of uterine discharge, in the other from a deficiency of renal, and we may infer in both capillary obstruction, with exudation as its accompaniment and arterial tension as its result.

We now come to hydræmia, theoretically distinct from anæmia, however associated with it clinically. Scantiness of urine is one of the most constant attendants of renal dropsy, though the companionship is not without exceptions. Urine may be totally suppressed, and that as the result of renal disease, without any trace of dropsy, as has been already pointed out; while in disease not renal—in diabetes, for example—dropsy may present itself while the urine is superabundant. Thus the quantity of the urine does not bear a perfectly simple relation to œdema, whether renal or diabetic, and so far as scantiness of urine may be taken to imply hydræmia, the complication extends itself to this condition.

Insufficiency of urine, other evacuations and drink remaining unaltered, must result in retention of water in the blood, increased fulness of vessels, and presumably increased filtration through their coats. Where this is not apparent, as with obstructive suppression, it is possible that the vessels are relieved by other evacuations, notably

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by vomiting. Taking all the facts together, considerable importance must be attached to hydræmia in connection with renal dropsy, though this condition *per se* does not appear sufficient to cause it. Cohnheim[1] has shown that blood diluted by nearly half its mass of a saline aqueous liquid may be passed through the vessels of a dog without any indication of œdema. I have already alluded[2] to a case of diabetic coma in which twenty-two pints of a watery solution were introduced into the circulation in thirty-two hours, with only traces of œdema and but little dropsy of any other kind. The legs, where the œdema was most marked, just above the ankle pitted only to the depth of an eighth of an inch. The small amount of dropsy which ensued upon this artificial and extravagant hydræmia appeared, as has already been shown, to be due rather to pressure than dilution; more to increase in the contents of the vessels than to their watery character.

So far as hydræmia is connected with renal dropsy, it may be inferred from the albuminous character of the effusions that the process is rather by filtration, or extrusion under pressure, than by dialysis.

The obvious insufficiency of the current explanations of renal dropsy have led to the substitution of theories which I can but briefly advert to. According to the high authority of Cohnheim, acute renal dropsy deserves the description which was given to it before Bright associated it with renal disease, when it was termed inflammatory dropsy, and looked upon as a general inflammation of the subcutaneous tissue. For my own part, I think that the general absence of the ordinary signs of inflammation of the cellular tissue, together with the character of the effused fluid, is sufficient to negative this view. Dr. Waymouth Reid has made a suggestion which he has not fully developed, and which perhaps is scarcely mature for discussion. He has been led to suppose that the dropsy of kidney disease is brought about by some process allied to

[1] Cohnheim's 'Pathology,' vol. iii, p. 1279.

[2] See p. 322.

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secretion on the part of the capillaries, and connected with "the protoplasmic activity" of their walls. He would thus regard the process as vital rather than physical; and would, I presume, look upon the effusion as vicarious, and the poisoned condition of blood as its excitant. If I am correct in thus presenting this view, it must at least be observed with regard to it that the vicarious secretion is often absent when the uræmic intoxication is most extreme, as with obstructive suppression, and sometimes with the advanced granular kidney. It is to be hoped that we shall hear more from Dr. Waymouth Reid on this matter. In the meanwhile it is certain that the dropsical process is not a perfectly simple one; if any such vicarious secretory action is concerned, it does not follow that other modes of action are to be excluded.

In acute renal dropsy (to limit attention for the present to this) the sphygmograph gives evidence of increased arterial tension, which is confirmed by the hypertrophy of the left ventricle.

Unless this arterial pressure is arrested short of the capillaries, we have a condition which must promote *exudation* from them; and the normal *absorption* also must be in many cases lessened by the inflammatory changes in the lungs, which with acute nephritis are the rule rather than the exception. But there are circumstances, among others the state of the right ventricle, which indicate that the part of the lungs and veins in renal dropsy is less important than that of the capillaries and arteries. It cannot be doubted that renal dropsy is essentially a matter of increased exudation, not diminished absorption, and that of this the capillaries are the seat. The process is connected probably with some change in themselves or in their relation to the blood, associated certainly with increased pressure behind them.

No argument is now necessary to connect the arterial tension and cardio-vascular hypertrophy with obstruction to the arterial exit. This must be either in the arterioles or the capillaries. If the obstruction is in the arterioles in the increasing dropsy of acute nephritis we are met with this

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difficulty, that the capillaries receive less but they discharge more, at least by the lateral exit; less blood enters them while more lymph leaves them.

It is well known that the arterioles have a power of muscular contraction which is used largely in obedience to the nerves, and which regulates the supply of blood to the various territories of nervous and vascular distribution. But this is not all. Bright thought that the capillaries presented a hindrance to the blood in his disease, and the progress of knowledge since has done much to enforce his conclusion. It was long ago shown by Poisseuille that liquids traversed capillary tubes with different degrees of facility according to the composition of the liquids.

In the living body the processes of inflammation were shown to involve changes of which the capillaries were the especial, and apparently the primary seat, which modified their diameter and otherwise affected the passage of blood through them, conceivably by giving adhesiveness to their walls. Though muscular fibres were not to be recognised in their coats, it was shown by exact experiment[1] that the capillaries were not only elastic, but possessed of vital contractility; thus variations in capillary resistance and in capillary diameter were placed beyond surmise. Whatever the influence of the nervous system upon the arterioles, great as it may be, we cannot but infer that it is upon the capillaries that must be exerted the direct influence of the blood in its character and by its contact. The coats of these vessels are penetrated by the exuding serum, while the blood maintains only a superficial contact with the arterial channels. Accepting as a probability that the capillaries are directly influenced by the uræmic blood so as to oppose a hindrance, be it of what sort it may, to its passage, we can understand the increase of tension, the increase of exudation, and something of renal dropsy.

Proceeding to chronic renal disease and the granular

[1] Paper by Drs. Roy and Graham Brown on "The Blood-pressure and its Variations in the Arterioles, Capillaries, and Smaller Veins," 'Journal of Physiology,' 1879-80, part 2, p. 323.

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kidney, we reach the apparent incongruity which has been stated in the diminution of dropsy with the further increase of arterial tension. With this association there is increased hypertrophy of the left ventricle, and thickening and narrowing of the arterioles. The inverse relation of the hypertrophy and the effusion, up to a certain point, is beyond question, however brought about.

The usual increase of urine must of course have its part in the process, but other conditions are certainly present, and must be taken account of. The thickening and constriction of the arterioles must cut off the blood from the capillaries, and presumably lessen pressure in them and exudation from them. The hypertrophy of the left ventricle with the increased thickness of its wall in relation to the cavity, as yet undilated, must give to this chamber an expansile power in diastole altogether beyond what is proper to it in health. The suction power thus increased must tend to draw upon the pulmonary circulation, and ultimately upon the veins and interstices, tending to pump the latter clear of any fluid which may have been effused. It is suggested that it is largely by this process, more or less of which would seem to be physically inevitable, that the antagonism between renal dropsy and cardiac hypertrophy is accomplished.

The giving way of the heart is the beginning of the end, and the return of dropsy its immediate result. The greatly hypertrophied ventricle, together with the mitral orifice, stretches, and mitral regurgitation occurs, enforced by a ventricular vigour unusual with this lesion. All the results of mitral regurgitation present themselves; pulmonary apoplexy frequently, and dropsy which may be truly called cardiac, though not cardiac *ab initio*.

Lardaceous disease presents itself as apart from the pathological series which has been traced. Increase of arterial tension and cardiac hypertrophy appear to have no concern with it. Excepting that the effusion varies more or less inversely with the amount of urine, it would not be safe to say more at present with regard to this result of

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the disorder than that it is different in its origin from other kinds of renal dropsy.

Putting this conclusion aside, the points of resemblance and of difference between renal dropsy and cardiac dropsy come into prominence. In both the accumulation, as is well known, is essentially, however modified, what exudes from the capillaries and constitutes the lymph. Under heart disease there is diminished absorption without any presumption of increased exudation, or not to any important extent. Under kidney disease there is increased exudation without any necessary or constant diminution of absorption. Both are essentially connected with modifications of blood-pressure,—in one from obvious obstruction to the venous return, in the other from some presumably obstructive condition of the capillaries which is attended with enhanced exudation from them, and associated with increased pressure within the arterial system.

It only remains for me to record my obligations to those from whom I have received help which I have not already acknowledged—to my colleagues without exception for unrestricted access to and permission to use their cases; to Dr. Delépine for aid in many ways beyond what has been already mentioned; to Dr. Rolleston in connection with the pathological aspects of the inquiry; and to a series of excellent and interested clerks, among whom I must name Mr. Pickthorne, Mr. Wilson, Mr. Thomson, Mr. Romer, Mr. Goldsmith, Mr. Little, Mr. Elliott, and Mr. Woodhouse for frequent and willing assistance.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 115.)

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NOTE.—All the sections shown in the forty-seven figures on pages 375 to 385 were reduced in the same proportion (nearly one third), so that correct comparisons can be made both as to size and shape.

DESCRIPTION OF FIGURES 1—8.

Heart in Health.

Sections of the heart in health made as described in the text. The exact forms and dimensions were reproduced by tracing, and ultimately reduced by photography.

This plate displays eight hearts in varying states of systole and diastole. All were free from valvular disease and apparently healthy, obtained from cases of suicide, accident, and brief illness. The ages of the subjects range from seventeen years upwards. The weights of the hearts, counting only adults, including both sexes, varied from 7 ounces to 14 ounces.

FIG. 1.—Male, aged 24. Weight 12 oz. Suicide.
FIG. 2.—Male, aged 28. Weight 13½ oz. Accident.
FIG. 3.—Male, aged 17. Weight 11 oz. Accident.
FIG. 4.—Male, aged 21. Weight 8 oz. Perforation of bowel.
FIG. 5.—Female, aged 52. Weight 14 oz, Acute bronchitis.
FIG. 6.—Female, aged 42. Weight 11 oz. Poison.
FIG. 7.—Female, aged 26. Weight 7 oz. Perforation of stomach.
FIG. 8.—Female, aged 17. Weight 6 oz. Perforation of bowel.

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FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.

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DESCRIPTION OF FIGURES 9—17.

Nine similar Sections of the Heart in Nephritis.

The last two cases, Kybert and Saunders, present decided hypertrophy, but as these were complicated with antecedent atrophy of one kidney, it is probable that the cardiac change was partially due to this cause. Some of the remaining cases show slight hypertrophy, none very much. With the boy Kelly the hypertrophy is decided; with the woman Grant there is hypertrophy together with dilatation. In all but one there was dropsy, generally considerable where the hypertrophy was slight.

FIG. 9.—Violet Ellwood, aged 9 months. Weight 5½ dr. No dropsy. Pulmonary apoplexy.

FIG. 10.—Florence Cox, aged 3½ years. Weight 2½ oz. Much dropsy.

FIG. 11.—John Kelly, aged 10 years. Weight 7 oz. Much dropsy.

FIG. 12.—John March, aged 12. Weight 6 oz. Much dropsy.

FIG. 13.—Maggie Grant, aged 29. Weight 14 oz. Much dropsy.

FIG. 14.—Maria Yockney, aged 50. Weight 10 oz. Much dropsy.

FIG. 15.—Arthur Maddison, aged 29. Weight 13½ oz. Slight dropsy.

FIG. 16.—James Kybert, aged 48. Weight 28 oz. Left kidney atrophied. Much dropsy.

FIG. 17.—John Saunders, aged 32. Weight 15 oz. Right kidney atrophied. Much dropsy.

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FIG. 9.

FIG. 10.

FIG. 11.

FIG. 12.

FIG. 13.

FIG. 14.

FIG. 15.

FIG. 16.

FIG. 17.

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DESCRIPTION OF FIGURES 18—27.

Granular Kidneys without Signs of Cardiac Failure.

Ten sections from old cases of the granular or fibrotic and contracted kidney, not conjoined with murmur or pulmonary apoplexy as signs of cardiac failure. The hypertrophy of the left ventricle is generally well marked, sometimes extreme; that of the right ventricle absent or slight, though in some instances it is to be recognised, notably in those of Weber and Fowler. The absence or slight measure of the dropsy in connection with the larger hearts is noticeable. Worby appears to present an exception; his case was one of nephritis and dropsy recently imposed upon an old fibrotic kidney. Fowler, Weber, Windover, Copcutt, and Crann illustrate the rule.

FIG. 18.—William Worby, aged 61. Weight 25 oz. General dropsy.

FIG. 19.—Peter Copcutt, aged 35. Weight 18 oz. Trace of œdema.

FIG. 20.—Emily Smith, aged 35. Weight 11½ oz. No dropsy.

FIG. 21.—George Windover, aged 58. Weight 22 oz. Slight œdema.

FIG. 22.—Henry Fowler, aged 43. Weight 23 oz. No œdema.

FIG. 23.—Charles Stocken, aged 58. Weight 13 oz. Slight œdema. Phthisis.

FIG. 24.—John Bartlett, aged 79. Weight 16 oz. Trace of œdema.

FIG. 25.—Edward Gooch, aged 44. Weight 10 oz. Dropsy.

FIG. 26.—Charles Weber, aged 64. Weight 16 oz. No dropsy.

FIG. 27.—William Crann, aged 47. Weight 26 oz. Trace of œdema.

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FIG. 18.

FIG. 19.

FIG. 20.

FIG. 21.

FIG. 22.

FIG. 23.

FIG. 24.

FIG. 25.

FIG. 26.

FIG. 27.

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DESCRIPTION OF FIGURES 28—33.

Granular Kidneys, with Signs of consequent Mitral Regurgitation; Valves natural.

Six sections from cases of the advanced granular kidney with signs of cardiac failure; five with pulmonary apoplexy, one with a mitral murmur only. The hypertrophy in all is great and dilatation for the most part evident, most so in the cases of Ruddick, Spaull, and Killick. There was much dropsy in every case but one.

FIG. 28.—Thomas Spaull, aged 24. Weight 18 oz. Much dropsy. Pulmonary apoplexy.

FIG. 29.—Walter Gurwood, aged 65. Weight 21 oz. Much dropsy. Pulmonary apoplexy.

FIG. 30.—Emma Killick, aged 53. Weight 17 oz. Much dropsy. Pulmonary apoplexy.

FIG. 31.—George Ruddick, aged 55. Weight 24 oz. No dropsy. Pulmonary apoplexy.

FIG. 32.—William Jones, aged 66. Weight 29 oz. Much dropsy. Mitral regurgitant murmur.

FIG. 33.—John Ford, aged 56. Weight 26 oz. Much dropsy. Pulmonary apoplexy.

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FIG. 28.

FIG. 29.

FIG. 30.

FIG. 31.

FIG. 32.

FIG. 33.

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DESCRIPTION OF FIGURES 34—39.

Lardaceous Disease of the Kidneys.

Six hearts from cases of lardaceous disease of the kidney. In one, Fyndley, nephritis has been recently superadded. The general weight is low compared with healthy hearts belonging to the same sex and age, the left ventricle generally weak with diminished muscular substance and increased cavity. The contrast between the heart of lardaceous disease and that of the granular kidney, in regard to the power of the ventricle, is striking. It is not necessary to draw attention to the obvious circumstance that the disease antecedent to the lardaceous was generally of a wasting kind. Dropsy was present in three of the six cases, in one due to superadded nephritis.

Fig. 34.—John Sedgewick, aged 47. Weight 8 oz. Phthisis. No dropsy.

Fig. 35.—Alma Hunt, aged 36. Weight 7 oz. Phthisis. Much œdema.

Fig. 36.—Patrick Fyndley, aged 34. Weight 8 oz. Syphilitic caries. Slight dropsy latterly.

Fig. 37.—Thomas Scales, aged 12. Weight 5 oz. Caries. No dropsy.

Fig. 38.—William Hall, aged 17. Weight 6 oz. Phthisis. Much dropsy.

Fig. 39.—Alfred Perrin, aged 23. Weight 12 oz. Bronchiectasis. No dropsy.

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FIG. 34.

FIG. 35.

FIG. 36.

FIG. 37.

FIG. 38.

FIG. 39.

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DESCRIPTION OF FIGURES 40—47.

A selection of Hearts in various Conditions of Disease for comparison.

It is seen that the left ventricle with the granular kidney nearly equals in bulk that belonging to any disease primary to the heart. The right ventricle, on the other hand, presents greater hypertrophy under cardiac disease or malformation than ever under kidney disease. The greatest hypertrophy of the right ventricle is shown in the case of Pearce (Fig. 47) as the result of congenital disease. The right ventricle here is absolutely thicker than the left. Considerable hypertrophy of the right ventricle is also shown in connection with mitral disease and emphysema (Cairns, Fig. 40).

FIG. 40.—Martin Cairns, aged 58. Weight 15½ oz. Mitral regurgitation, emphysema, and bronchitis. Much dropsy.

FIG. 41.—Edwin Rose, aged 36. Weight 24 oz. Pericardial adhesions. Bronchitis. Œdema.

FIG. 42.—Louisa Jones, aged 21. Weight 10 oz. Fatty degeneration. Anæmia. Œdema of feet.

FIG. 43.—Walter Banks, aged 28. Weight 33 oz. Aortic and mitral valves diseased. Little dropsy.

FIG. 44.—Maria Ware, aged 32. Weight 8 oz. Phthisis. No dropsy.

FIG. 45.—Georgina Hough, aged 50. Weight 27 oz. Mitral regurgitation. General dropsy.

FIG. 46.—George Paulger, aged 60. Weight 26 oz. Aortic stenosis. Œdema.

FIG. 47.—William Pearce, aged 19. Weight 20 oz. Congenital pulmonary stenosis. Perforation of septum ventriculorum. Phthisis. Slight œdema. The ventricle placed uppermost in the diagram is the right; it is seen to have absolutely thicker walls than the left.

N.B.—The thin line which appears on the outside of some of the hearts shows the amount of fat upon them.

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FIG. 40.

FIG. 41.

FIG. 42.

FIG. 43.

FIG. 44.

FIG. 46.

FIG. 45.

FIG. 47.

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NOTE

ON THE

ALLEGED OCCURRENCE OF OVARIAN PREGNANCY;

BEING AN

EXPLANATION OF SOME CASES WHICH HAVE BEEN PUBLISHED AS BELONGING TO THIS VARIETY OF ECTOPIC GESTATION.

BY

LAWSON TAIT, L.R.C.P.Ed.,

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Received February 11th—Read June 14th, 1892.

In the scheme of ectopic gestation which I have published on page 8 of my book on 'Ectopic Pregnancy and Pelvic Hæmatocele,' the first variety in order of narration is that of the so-called "ovarian pregnancy," concerning which I say that it "*is possible, but not yet proved.*" I could point to at least half a dozen critics who, perfectly regardless of this clear enunciation of opinion, say that I have denied the possibility of its occurrence; and then they proceed to demolish the views which they have manufactured for me. On page 13 I say, "It is possible, I admit; but there are so many contingencies in such a case, that the doctrine of chances makes it so remote that its occurrence may be regarded as likely as the birth of a blue lion or a swan with two

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necks, like an heraldic monstrosity—a mere pathological curiosity. Finally, it will have no kind of clinical interest or importance not already decided upon in the case of pregnancies in the free part of the tube, so that we may avoid any further discussion of ovarian pregnancy as futile. If it does occur, it must be rare and will be curious. If it never occurs, so much the better."

Since the publication of my book several specific efforts have been made to prove from recorded instances and preparations exhibited that a real ovarian pregnancy has occurred, and about these instances I have a final word or two to say; and after I have said that, I can only feel that further criticism against such assertions must be futile: for if in the discussion of the subject, which has gone on now for over three years, better examples than those I speak of cannot be adduced, I think we may accept the conclusion that the occurrence of ovarian pregnancy is very unlikely.

In a true ovarian pregnancy the following conditions must be present:—The gestation sac must be formed entirely of ovarian tissue, so that in all parts of the sac wall ovarian elements can be discovered by microscopic research. The tube must be free in its entirety from the gestation sac, taking no part in its formation. The broad ligament also must be free, forming merely the pedicle of the tumour, and not having the tumour embedded between its layers. I hold that there is a possibility of such an occurrence. But to produce it the Fallopian tube would have to be patent throughout, and deprived of its lining epithelium by desquamative salpingitis. The abdominal end of the tube would have to be unoccluded. A Graafian follicle would have to rupture in such a fashion as to permit of the ingress into its cavity of the impregnating spermatozoa (thus to secure its fertilisation), and yet to prevent the egress of the ovum from its follicle after fertilisation. To complete this great chain of improbabilities, the impregnated ovum would have to become adherent to the wall of the follicle

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and develop in its cavity. The unlikelihood of the occurrence of this last condition is greatly enhanced by the recollection of the fact that the follicular wall contains no adenoid tissue such as seems to be essential for the nourishment of a recently fertilised ovum. It may safely be asserted that no case has yet been brought forward which has been submitted to these tests and subsequently been established as a true ovarian pregnancy.

It is easy to see, however, how inexperienced observers have been led to describe as ovarian pregnancies cases of broad ligament pregnancies. They rely on two signs—(1) the presence of ovarian elements in some part of the gestation sac wall; (2) the apparent absence of the ovary on that side. But these signs are capable of another and much more reasonable explanation indicating the true nature of the case; whilst the explanation usually given of them, that they are indicative of true ovarian pregnancy, is wholly deceptive and misleading. I ask you to consider the changes in the anatomical relation of the various constituents of the broad ligament produced by a tumour developing between its layers and becoming embedded therein. These alterations of relations are brought about by a process which is made perfectly familiar to us in the ordinary phenomena of pregnancy, and other conditions in which the peritoneum is slowly moved about by traction.

Mr. Christopher Martin has devised some diagrams which illustrate my point. Fig. 1 is a diagram of a lateral vertical section through a normal broad ligament. The Fallopian tube is uppermost, and is surrounded on all sides save below by peritoneum, which slides freely over it. The ovary is attached to the posterior layer of the broad ligament, and at the line of junction the cubical epithelium covering the ovary becomes continuous with the superficial epithelium of the broad ligament. Observe that the peritoneum does not pass over the ovary as a freely sliding membrane, but is continuous with its fibrous investment. Hence if there be a constant and prolonged

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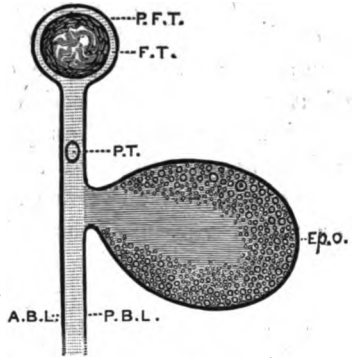

FIG. 1.—Diagram of lateral vertical section of broad ligament.

F.T. Fallopian tube. P.F.T. Peritoneum covering Fallopian tube. P.T. Parovarian tubule. Ep.O. Epithelium covering ovary. A.B.L. Anterior layer of broad ligament. P.B.L. Posterior layer of broad ligament.

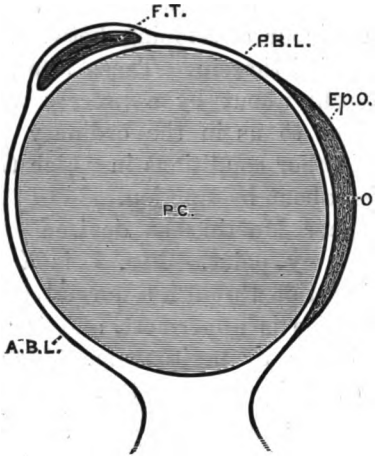

FIG. 2.—Lateral vertical section of broad ligament with embedded parovarian cyst.

F.T. Fallopian tube. P.C. Parovarian cyst. O. Ovary. Ep.O. Epithelium covering ovary. A.B.L. Anterior layer of broad ligament. P.B.L. Posterior layer of broad ligament.

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strain, such as is produced by the separation of the two layers of the broad ligament by a tumour developing between the two layers, both layers are put tensely on the stretch. In consequence of this, a strain or pull is exerted on the ovary itself, and this strain is, for practical purposes, directed equally round its edge. The result of this is shown in Fig. 2. Here we see that one of the parovarian tubules (shown in Fig. 1 as a small vesicle) has become enlarged into an intra-ligamentous cyst. It has developed between the two layers of the broad ligament, and has consequently put the peritoneum of the broad ligament greatly on the stretch. In so doing it has altered the relation of parts. The anterior layer is widely separated from the posterior. Everywhere the peritoneum is distinct from the cyst proper, and can be peeled off from it. The Fallopian tube is seen to be flattened and widened by the traction due to the growth of the cyst. It is, however, easily separated from it by raising the peritoneum. One half of the circumference of the tube is deprived of its peritoneal covering. But specially observe the condition of the ovary, which has become flattened and greatly extended towards its periphery. It has been pulled out and spread out into a thin layer of ovarian tissue, the traction having opened the ovary out into a sheet, starting at the hilum. The extended ovary is, however, easily separable from the cyst proper by raising the peritoneum, all the ovarian tissue rising with the peritoneal layer. Do not misunderstand me; this does not occur in all, or even in a majority of embedded parovarian cysts; but every now and then it is to be met with in a very marked degree. An actual specimen is now exhibited where the right ovary has been spread by the growth of a parovarian cyst into a circular sheet five inches in diameter. Fig. 3 represents a broad ligament pregnancy. It was originally tubal. The tube has ruptured in a downward direction into the potential space between the two layers of the broad ligament. The ovum has developed in the

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broad ligament in a manner closely resembling that of an embedded parovarian cyst. In so doing it has disturbed

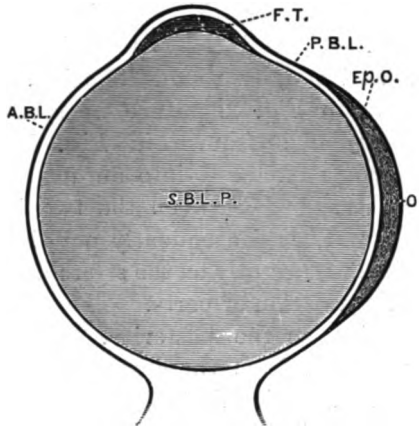

FIG. 3.—Diagrammatic representation of broad ligament pregnancy. F.T. Fallopian tube. S.B.L.P. Sac of broad ligament pregnancy. O. Ovary. Ep.O. Epithelium covering ovary. A.B.L. Anterior layer of broad ligament.

the anatomical relations of the parts in precisely the same way. The two layers of the broad ligament are widely separated and put on the stretch. The Fallopian tube has become spread out over the top of the ovum as a sheet of muscular tissue. The ovary has likewise been flattened out, or rather pulled out, into a sheet of ovarian tissue, lying in close contact with the gestation mass.[1]

[1] In the growth of parovarian cysts it is to be noticed that the Fallopian tube is usually not much flattened, the reason of this being that its peritoneal covering, unlike that of the ovary, is easily stripped off. But it may be enormously lengthened by the traction. I have seen it lengthened as much as fifteen inches; Bantock records an elongation of twelve inches; and a specimen is now shown, from a parovarian cyst removed by Mr. Christopher Martin, in which it was thus elongated to ten inches. This process of the alteration of structures so important as the ovary and Fallopian tube by mere mechanical stretching in the process of the shifting of the peritoneum, necessitated by the pathological growth of structures included in its folds, has not previously been prominently noticed. Doubtless its influence will now be

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So greatly is it extended and altered that an inexperienced observer might easily miss it, and thus record that "the ovary could not be found"—a statement which is constantly made in such cases, and which, quite true in itself, is assumed to prove that the seat of the gestation is the ovary itself, whereas it really is the broad ligament, upon the outer surface of which the ovary has been pulled out into a transparent sheet.

It is thus very easy to understand how it is that ovarian tissue is found in the sac wall by microscopic research if the section happens to fall within the area of the flattened-out ovary. And, curiously enough, it is always noted in such cases that the ovarian tissue is not to be found uniformly distributed over the wall of the cyst. A careful consideration of these points will explain how it is that a broad ligament pregnancy has been so often erroneously described as a "true ovarian pregnancy."

discovered in other instances. The only record of its observation, so far as I know, is a somewhat casual one by Mr. Alban Doran in his book on 'Tumours of the Ovary,' 1884.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 151.)

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UPON

AMPUTATION IN DIABETES MELLITUS.

BY

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Received March 4th—Read June 14th, 1892.

Such stress has been laid upon the danger from inflammatory complications in Diabetes Mellitus, that many writers condemn operative procedure without qualification, or at least omit to state the indications for it. Yet there is enough evidence, I believe, to show that, by the timely adoption of major amputations, life may be prolonged and the amount of sugar excreted reduced to a minimum in cases where the attempt to effect this by medicinal treatment, in the presence of a severe inflammatory lesion, has quite failed.

Many inflammatory lesions heal readily under the general treatment for diabetes combined with local antiseptic measures, such as boils, carbuncles, and gangrenous ulcerations of the penis. The following case shows that there is no want of repair in such patients.

Case 1.—A carman, æt. 24 years, had suffered before coming under my care from increased frequency of micturition, excoriations on the penis which became gangrenous ulcers, and an abscess in the left groin which was incised,

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the case being taken to be venereal. I could find no evidence of venereal infection, but, on the other hand, there was a large amount of sugar in the urine. The whole of the left lower extremity and scrotum were swollen, red, and painful from cutaneous erysipelas, whilst the sinus resulting from the incised abscess was discharging thin foul pus. After admission to the Westminster Hospital the inflammation in the leg subsided, and the ulcers upon the penis as well as the sinus in the groin healed. The urine was 1032 in specific gravity and 150 to 200 oz. (4·25 to 5·67 litres) in amount, the sugar being 8 grs. to the oz. (1·8 grms. per 100 c.c.). He took codeia and a full diet from which starch was withdrawn. Three weeks after his discharge, whilst upon the same medicine and diet, he died in diabetic coma. This fatal termination proves that the patient was suffering from a malignant form of diabetes, but one which did not prevent his recovery from an acute attack of erysipelas less that six weeks before the end.

The two following cases under my care, very similar in character, illustrate the failure to treat in the one case, and the success following amputation of a diseased limb in the other.

Case 2.—A hairdresser, æt. 51, was admitted to the Westminster Hospital on September 7th, 1887, with a wound on the inner side of the left knee. A week before he was using a chisel, when the instrument slipped, and the blow drove it into the left leg on the inner side of the patella. The small wound bled slightly, and he covered it with some rag. After two days his knee became painful. For two years he had suffered from excessive thirst and increased frequency of micturition. He had also drunk hard. The lower part of the thigh and knee were obviously swollen, and when pressure was made upon the patella pus spurted from the wound. Temperature 100·4° F. Urine acid, 1040; no albumen; a large quantity of sugar. The

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wound was found to be the orifice of a large cavity placed on the inner side of the knee, which communicated with the joint and with another abscess cavity beneath the quadriceps extensor tendon. The cavities were drained and constantly washed out. Bread and potatoes were withdrawn from an otherwise full diet, codeia, ammonia, and bark being also administered.

For the next week his temperature varied from 99° F. to 100° F.; the amount of urine was about 100 oz. per diem (2·8 litres), containing about 20 grs. to the oz. (4·4 grms. per 100 c.c.). Drainage was good, but it was obvious that his life could only be saved by amputation. I then believed that amputation in diabetes mellitus was sure to be followed by gangrene of the stump and death.

He died on September 20th in diabetic coma, the temperature rising to 104° F., and the breath becoming sweet.

At the post-mortem the cadaver had a well-marked sweet odour, the kidneys were slightly granular, the abscess cavities almost surrounded the lower third of the femur, but there was no collection of pus, nor any bone bare. The arteries and veins of the limb as well as the rest of the body were quite healthy.

I fortunately treated the following very similar case in a better way.

Case 3.—A tailor, æt. 56, had for some time suffered from a dry sore mouth, from an irritable skin, and great thirst. He had formerly drunk spirits freely, but latterly four pints of beer daily. On August 29th, 1892, he felt pain over the thenar eminence which gradually extended to the left arm. On September 1st he first noticed a swelling behind the elbow, and was admitted to Westminster Hospital on September 2nd. He could not move his arm, which was swollen from above the elbow to the middle of forearm, being about double the size of the right, was hot, and of a dusky red colour. The olecranon bursa was distended, and a quantity of blood-stained serous fluid escaped

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on incision. There was much sugar and a trace of albumen in the urine.

He was placed upon a full diet excluding bread, potatoes, and sugar. Opium was administered, and the arm kept in a boric acid arm-bath. The swelling of the arm did not go down. The temperature rose to 101°—102° F. at night. The urine passed was about 70 oz. (2 litres), sp. gr. 1035—1042, containing about 10 grs. to the oz. (2·2 grms. per 100 c.c.). On September 8th I extended the bursal incision upwards above the olecranon through the triceps. A quantity of pus escaped, and the lower third of the posterior surface of the humerus was found to be bare. The pus had also burrowed into the subcutaneous tissue, but the joint had practically escaped.

I urged the patient on the next day to submit to amputation, but he steadily refused. Although all care was taken of the drainage, the suppuration became daily more profuse, and the sugar excreted was quite uninfluenced by the medicinal and dietary treatment. On September 21st the patient had become prostrate, semi-comatose, with incontinence of urine and fæces, and then the patient's friends consented to the amputation. This I did on September 22nd, amputating the left arm at the level of the attachment of the deltoid. Unfortunately this was below a small line of inflammatory induration which had extended up along the course of the basilic vein to the level of the coracoid process. Owing to the delay I could not have obtained flaps completely free from inflammation even by the more severe operation through the shoulder-joint. The artery was quite healthy. The stump itself appeared to heal by first intention, but the inflammation along the basilic vein formed a superficial abscess which required incision, and later on a counter opening had to be made to finally cure a sinus which persisted. He went to a convalescent home in the middle of December, and returned in January with the stump healed, feeling well, and having gained much weight. The result of the operation upon the temperature was that it become subnormal for a fort-

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night, then normal. The specific gravity of the urine fell from 1036 on the day of operation to 1023, then gradually rose during the development of the abscess, remained at 1030 during the persistence of the sinus, and has finally fallen to 1023. The sugar fell from 10 grs. to 2 grs. to the ounce, then rose to 8 grs., and finally returned to 1½ grs. During recovery opium and codeia appeared of no service, and were omitted; but after healing of the stump, codeia being again given, the sugar was reduced to a well-marked trace (*vide* Chart).

This case shows that the amount of sugar excreted by a chronic diabetic is greatly increased by an inflammatory affection, and it illustrates the inefficacy of the drug and diet treatment. Had the operation been done earlier, I believe that the wound would have healed by the first intention, and the sugar excreted have fallen to a minimum at once.

The severest inflammatory complication in cases of diabetes mellitus is that of gangrene, but I would submit that there is good evidence to show that the sugar in the urine is not the cardinal factor upon which the plan of treatment ought to turn, but is only one of several. The gangrene scarcely ever occurs in patients below forty years of age; it is absent in the more malignant forms of diabetes, and it nearly always appears in chronic diabetics who present along with the gangrene an exacerbation of the glycosuria. A large majority of such cases have arterial sclerosis, affecting especially the vessels of the leg, and the gangrene commences like senile gangrene. More rarely the nerves are affected, and the first lesion is a perforating ulcer.

Again, many cases present conditions of impaired nutrition due to gout, alcohol, or chronic renal disease.

I would, therefore, urge that a consideration of the recorded cases leads to the conclusion that the failure of amputation for diabetic gangrene has been due to one or more of the following causes:

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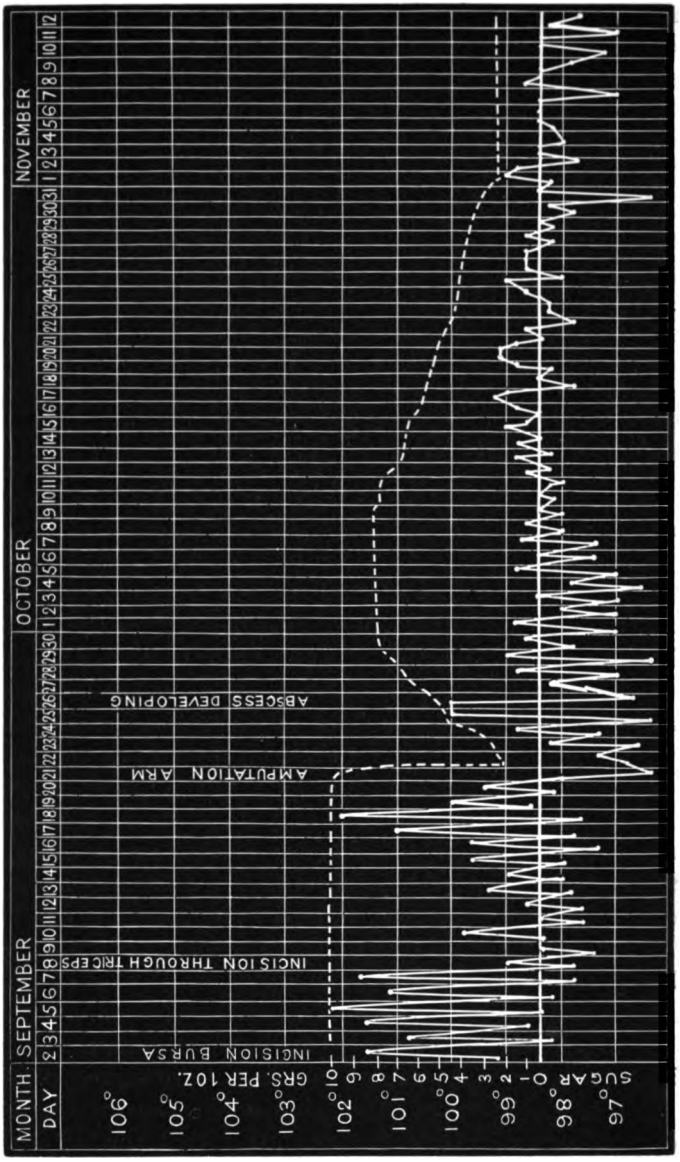

The dotted line - - - - - indicates the amount of sugar in the urine. The thick line is the temperature chart.

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1. Amputating through the leg when the arteries have already become occluded.

2. Amputating through inflamed tissue, or by septic inoculation, formerly, of the wound at the operation.

3. Postponement of the operation till too late, especially after the urine has become albuminous.

Of these I must lay most stress upon the failure from operating too low. Success has been obtained when the above causes have been excluded. Thus Mr. Hutchinson has recorded one case in his important paper in the volume of the Society's 'Transactions' on high amputation for senile gangrene. Other cases have been recorded by Professors Roser and König, who have for a long time been in favour of amputation; also by Kraske. But the most important paper has only lately come to my notice by Heidenhain, giving an account of Professor Küster's operations. Twenty-five cases of senile and diabetic gangrene were treated by amputation, all of the lower limbs. Fourteen were cases of senile gangrene only, and the mortality five. Eleven were in diabetics, and the mortality was also five. But if we look nearer at the five fatalities in diabetes mellitus, this result is seen not to militate against operation. One was in diabetic coma, at the time with 5 per cent. sugar; death took place in twenty-four hours, the coma persisting. The second case underwent amputation through both thighs at a week's interval, having 8 per cent. of sugar. He died at the end of nine days from sudden pulmonary œdema and coma. Both stumps were in course of healing by first intention. A third case also underwent a double amputation through the thigh at three months' interval. At the second operation he had 1·3 per cent. sugar and one fifth albumen. He died after five days in coma, the stump being in process of healing by first intention. The fourth case had much sugar and two thirds albumen; he also died after five days, the amputation wound through the thigh tending to heal without suppuration. The fifth case was reported to be a diabetic, but had no sugar

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before nor after the operation, only much albumen; he died on the fifty-eighth day, after amputation through the thigh, of cardiac failure. The diabetes in these cases appears not to have affected the healing of the wound. In the three cases dying in diabetic coma, after five and nine days, the stumps were healing by first intention. In three of the cases albumen was present in large amount. Of the six recoveries after amputation through the thigh two healed by first intention, the other four after more or less ulceration.

The sugar before operation was (1) "large;" (2) 5 per cent.; (3) 3·6 per cent. with one third albumen; (4) 2 per cent.; (5) 1 per cent.; (6) a trace, and fell very much in all the cases after operation.

A most important point in the paper is the evidence in favour of high amputation. Of the twenty-five cases twenty had arterio-sclerosis, and in eleven of these the vessels of the leg were almost completely occluded by thrombi.

Professor Küster amputated low down in some of the earlier cases. In four cases a toe was removed, which was followed by gangrene of the foot. One case died of sepsis after a secondary Lisfranc (1871), one recovered after a secondary amputation through the leg and a tertiary amputation through the thigh. The other two recovered after secondary amputation through the thigh.

Three cases of Chopart's amputation were likewise followed by gangrene, and all three recovered after a secondary amputation through the thigh.

Of six amputations through the leg, one died of gangrene and sepsis. Two healed after limited ulceration and necrosis, and three recovered after a secondary amputation through the thigh. Two cases were amputated through the knee-joint; one healed by first intention, one after secondary amputation through the thigh. The long anterior flap of this operation is so likely to fail if there is any arterial narrowing.

I have only met with the account of three cases of

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amputation through the leg in diabetics which were successful, all three after some ulceration and necrosis. König had two cases, the sugar being 4 per cent. and 2·22 per cent., which after healing had become a trace. One was under Mr. Langton at St. Bartholomew's, which he has kindly allowed me to mention.

Case 4.—A carpenter, æt. 65, the subject of gout and addicted to alcohol, began to suffer, May, 1884, from gangrene of the toes of the left foot. His arteries were markedly calcareous, distinct rings being felt in the radial vessels. The urine varied between three and four pints (2 litres), containing five grains to the ounce (1·1 gramme per 100 c.c.) of sugar, and some albumen. The leg was amputated through the lower third on July 31st; the vessels were very calcareous, and the stump was finally healed in April, 1885, after limited ulceration and necrosis. The recovery was complicated by the occurrence of boils, carbuncle, and by local gangrene of the opposite foot. On his discharge the urine contained only a trace of sugar and no albumen.

Primary union, therefore, can only be expected after a high operation.

Diabetic gangrene, whilst still limited to a toe, can often be arrested, as in the case of a woman under my care, who had suffered from diabetes mellitus for more than five years before gangrene occurred for the first time. It recurred twice, in the same toe on all three occasions, yielding to treatment, but I have not met with any case of recovery when the deeper structures of the foot have become involved. A line of demarcation may form round the base of the toe and yet prove deceptive, because the gangrenous process continues to burrow insidiously along planes of fascia and tendon-sheaths. Sir James Paget has emphasised this point, and I have observed the same thing.

Perforating ulcers, however, offer a more favorable prognosis, be it that the discharge can be prevented from

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accumulating, or that the vessels remain relatively sound. I have had one case of extensive ulcers which healed, and Mr. Thomas Smith mentioned another to me in which the foot appeared to be bored through by several sinuses, and yet healing finally took place.

In all the cases referred to in this communication amputation was performed because a fatal issue was imminent, so that the recoveries represent so many lives prolonged. The amount of sugar before operation and its existence after, although much reduced in quantity, establishes the existence of diabetes mellitus in them.

And I believe that similar successes will be obtained in the future—(1) by adopting a high amputation; (2) by avoiding the delay involved in the attempt to reduce the sugar by drugs and diet in the face of a severe inflammatory complication.

To put the conclusions in another form, diabetic gangrene should be operated upon by following the rules already established for other forms of gangrene.

Note (June, 1892).—The patient continues in good general health, and was shown at the meeting; the stump has quite healed. Since becoming an out-patient the diet has not been so well adhered to, and the sugar in the urine has varied. The sp. gr. was on one occasion 1032, on another 1019.

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TWO CASES

OF

PYLORECTOMY AND ONE OF JEJUNOSTOMY,

WITH REMARKS,

AND WITH A SUGGESTED MODIFICATION OF THE FORMER OPERATION.

BY

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PROFESSOR OF SURGERY IN THE VICTORIA UNIVERSITY AND HON. SURGEON IN THE LEEDS GENERAL INFIRMARY.

Received January 5th—Read June 14th, 1892.

I AM bringing forward the following cases for two reasons: 1st, because it seems to me important that all serious and especially comparatively new operative procedures should be chronicled for statistical and comparative purposes; 2nd, because it is not yet clearly established that these operations for cancer can be relied on to produce such an amount of relief as to warrant their continuance.

Concerning the operation of gastrostomy for the prolongation of life in impermeable cancer of the œsophagus, and gastro-enterostomy for pyloric obstruction, I think there is sufficient evidence to prove that the relief

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afforded is amply sufficient to warrant the continuance of their practice, and I will not spend the time of the Society by detailing cases, as they have been discussed fully here and elsewhere; and, moreover, I have myself written on both subjects in the 'British Medical Journal' for June 7th, 1890, and the 'Lancet' for May 23rd, 1891. I think, however, that I ought to mention a sequence of gastro-enterostomy which has twice occurred in my practice, and which, so far as I am aware, has not been previously mentioned; it is the regurgitation of the intestinal contents into the stomach through the artificial opening, interfering with assimilation and producing offensive eructation and nausea. The three following cases are given in detail:

Case 1. *Pylorectomy for pyloric stenosis due to cancer; recovery; recurrence of symptoms due to contraction of the cicatrix; gastro-enterostomy four months after; regurgitation of intestinal contents and fæcal poisoning.*—W. S—, æt. 54, a cabinet-maker, was admitted to the Infirmary on June 23rd, 1891, when he gave the history that he had been quite well up to six months previously, when he began to lose his appetite and to experience a feeling of weight at the stomach after food. This was soon followed by pain in the epigastrium, with occasional vomiting of the stomach contents. During the past two months the vomit had been dark-coloured, and had occurred almost constantly after food. Beyond a general fulness of the abdomen he had not noticed any localised swelling. During the eight months he had lost about two stone in weight.

On admission the patient was found to be much emaciated, and on examination of the abdomen well-marked signs of distension of the stomach were discovered. A tumour about the size of a walnut could be felt an inch to the right of the middle line and an inch and a half above the umbilicus; this was rather painful on pressure, and at some times was much more distinctly felt than at others.

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The tumour, which was dull on percussion, was not fixed, and moved up and down with respiration.

Vomiting occurred after every meal, the vomit consisting of almost unaltered food mixed with coffee-ground material, and on examination blood was found to be present and free hydrochloric acid absent. A diagnosis of cancer of the pylorus was made, and pylorectomy was decided on after consultation.

Liquid food only was administered on June 24th, and after dinner all food was stopped by the mouth, nutrient injections being given. In the evening the upper part of the abdomen was shaved, an antiseptic dressing applied, and the stomach washed out with a solution of boroglyceride; the stomach lavage was repeated on the morning of the 25th, and again half an hour before the operation, which was performed at 2 p.m. on June 25th, the A. C. E. mixture being the anæsthetic.

An incision of three inches was made in the linea alba above the umbilicus, and after a few peritoneal adhesions had been broken down, the pyloric tumour was easily brought to the surface; the disease was apparently limited to the pylorus, extending only a small distance along the anterior wall of the stomach. A handled needle with a slit on its convex side was pushed through the gastro-hepatic omentum above the pylorus; the loop of a silk ligature was introduced into the slit and the needle withdrawn, thus leaving, after division of the loop, two ligatures *in situ*, which were then tightened, the gastro-hepatic omentum being divided between the two; this process was repeated on the right and left of the first ligatures, thus separating the pylorus above. The index finger of the left hand was then passed through this opening into the lesser cavity of the peritoneum, and a similar procedure was adopted in the division of the great omentum, no blood being lost in the process.

Hahn's clamps were then applied to the stomach and duodenum beyond the disease, which was then removed by means of scissors. After all bleeding points had been

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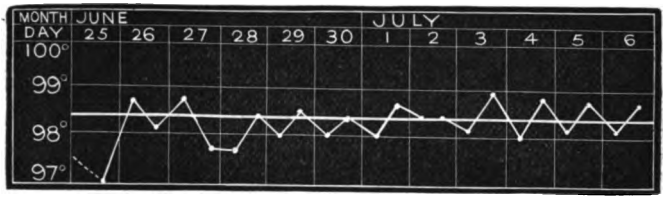
ligatured, the wound in the stomach was closed by a continuous suture, and made secure by Lembert's sutures from above downwards, leaving an opening at the lower end equal in size to the opening in the duodenum.

The peritoneum of the posterior halves of the openings in the stomach and duodenum was first brought together by Lembert's sutures; the mucous membrane of the stomach and duodenum were then connected all round, and lastly, the anterior halves of the stomach and duodenal openings were connected by Lembert's sutures. As sponges had been packed around the site of operation, the peritoneum had not become in any way fouled, and practically no blood was lost. The parietal wound was then closed in the ordinary way by six silkworm gut sutures. The whole operation occupied an hour and a quarter. The after progress was all that could be desired, the temperature never exceeding the normal, and no shock being



manifested. During the first twenty-four hours there was a little vomiting of dark-coloured fluid; on the second day Brand's essence and barley water were given; on the third day milk and soda water and beef tea were administered; on the fourth day the patient took about three pints of milk, with Brand's essence and other light food.

On July 4th, *i. e.* a week after the operation, the wound was dressed for the first time and found to be healed, the stitches being removed. The patient was then taking fish and rice pudding; the next day he had chicken; and the following day he took a mutton chop with good relish, and digested it. He rapidly regained strength and flesh,

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and was made an out-patient within a month of the operation, expressing himself and feeling and looking well.

The tumour removed was about the size of a large walnut, and hard both to touch and on section. The peritoneal surface was smooth and apparently unaffected.

A clear margin of healthy tissue intervened between the growth and the cut surface.

The mucous surface was dark and ulcerated.

The pyloric orifice was much constricted, admitting when relaxed the tip of the little finger.

August 21st, 1891.—The patient came to the infirmary to report that his vomiting had returned, and that he was losing the flesh he had regained. The vomit consisted of fermented food, and was entirely free from the coffee-ground character which had previously been present. It contained stringy mucus and yeast-cells, but no sarcinæ and no free hydrochloric acid; the vomiting came on half an hour to two hours after food. There were manifest signs of dilatation of the stomach, but no tumour or other abnormality could be felt. Although the stomach was washed out daily he rapidly lost flesh, and on September 8th a note was made to the effect that he had lost nine and a half pounds since admission. On September 9th it was manifest the patient was rapidly running down, and he was so weak that it was felt inadvisable to continue the lavage, and questionable whether he was strong enough to bear any further operative procedure; but as it was now evident that the pyloric opening had again contracted the state of affairs was explained to him and his wife, and the operation of gastro-enterostomy was proposed.

On September 10th, ether being the anæsthetic, the abdomen was opened in the line of the old scar. So far as could be discovered there was no return of the disease at the pylorus, and the parts appeared quite natural except for the dilatation of the stomach. A loop of the jejunum was easily found by following it down from its fixed point on the left of the spine; this was then emptied and rendered bloodless by drawing it between the fingers of the

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left hand, and encircling its base by a tourniquet composed of a piece of elastic tubing, which was tied in a single knot and clamped by pressure forceps. Senn's bone-plates were then introduced through incisions in the bowel and the lower part of the stomach, and were fixed in the usual way, four Lembert's sutures being introduced around the plates for greater security.

The peritoneum was not soiled in any way, and the abdomen was closed by silkworm gut sutures. The operation occupied three quarters of an hour. The patient rallied well from the operation, although in the evening his temperature was only 97·2°.

The nutrient injections were continued every four hours, and a teaspoonful of Valentine's meat juice was given every hour. On September 11th the patient seemed better, and had neither vomiting nor pain, but the meat juice had to be stopped for a few hours, as it made him sickly. Temperature normal. Pulse weak. On September 12th, beyond the weakness, the patient seemed fairly well, being entirely free from pain or sickness. His breath, however, had an unpleasant odour, and the nutrient enemata were not retained, but brandy and liquid food was given by the mouth and retained. On September 13th, without any new symptoms developing, the patient became gradually weaker, and died exhausted in the afternoon. I have to thank my colleague Dr. Barrs, Honorary Pathologist, for the following report:

Post-mortem examination made September 14th:—"Extremely emaciated body, scarcely a particle of fat anywhere, slaty-black staining of peritoneal surfaces, probably due to blood-staining of operation in June. No general peritonitis. Some old adhesions in pyloric region. Recent adhesion of loop of jejunum to pyloric end of stomach, embedding Lembert's sutures. It may be said the operation of gastro-enterostomy had been quite satisfactory, the highest part of the jejunum available had been opened into the lowest possible point of the stomach,

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and everything was in good order. The intestinal plate was partly eroded, the stomach plate was loosened and softened, and the ligatures of both were still *in situ*. There was a patent and sufficiently capacious opening between the two viscera. The stomach contained some grumous, fæcal-looking fluid, and the mucous membrane was stained in such a manner as to strongly suggest that the intestinal contents had passed into the stomach. The gastric musculosa and mucosa were much hypertrophied. The pyloric orifice would not admit the tip of the little finger without force, the natural pyloric ring being replaced by a sharp, tight, cord-like cicatrix. Externally the parts had an almost natural appearance, and there was no sign of new growth in the stomach or in any other organs. All the other organs were healthy.

Case 2.—Mr. T. B—, æt. 57, called to consult me on the 10th of June, 1891, on account of dyspeptic symptoms accompanied by vomiting and great loss of flesh. He told me that he had been well up to October, 1890, when he began to have discomfort at the pit of the stomach, with a want of appetite and sickness, but no vomiting since Christmas. He had been treated for indigestion, and had been taking medicines of various kinds without benefit, and during the six months previous to my seeing him he had lost 22 lbs. in weight. During May and June he had vomited every second or third day. When I saw him there were manifest signs of dilatation of the stomach, and a tumour about the size of a hen's egg could be felt midway between the umbilicus and the right costal margin. His immediate symptoms were relieved by washing out the stomach, and by the administration of salicylate of soda and strychnia. The pain disappeared, the vomiting ceased, and he was less troubled with flatulency and constipation. For a fortnight he ceased to lose weight, but without any corresponding increase in strength. My diagnosis was cancer of the pylorus, and this I explained to Mr. B—

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and his friends, telling him at the time that I saw no chance of relief except by operation.

On July 2nd Dr. Barrs saw the patient with me, and agreed as to the advisability of operating.

On July 7th pylorectomy was performed as in the last case, but as the tumour extended for some distance along the duodenum the resection was more extensive, and it was necessary to remove an extra portion from the posterior wall of the duodenum, as it was found that the section was made quite close to the growth. Two enlarged glands were also removed. No special difficulty was experienced in bringing the parts together, and very little blood was lost. The operation occupied one hour and a half. The patient rallied well, experienced very little shock, and only vomited once. Barley water in teaspoonful doses was given during the first twenty-four hours, after which Valentine's meat juice, Brand's essence, and peptonised milk and soda water with a little brandy were given. Nutrient injections were also administered, but unfortunately were never retained.

The patient appeared to be doing fairly well up to the third day, except that he did not regain strength, and from the evening of the third day until the termination of the case he simply gradually got weaker, and died of exhaustion on the morning of the fifth day.

So far as could be ascertained in the absence of a post-mortem examination, death appeared to be due simply to exhaustion, and not to any complication of the operation, as there was an entire absence of distension, vomiting, or other sign of peritonitis. The resected tumour proved to be carcinoma, which had almost obliterated the lumen of the pylorus, so that it would only admit the passage of a No. 4 catheter.

Case 3. *Extensive cancer of stomach, with inability to retain food; jejunostomy; recovery from operation; death from progress of disease at end of two months.*—Mrs. E. B—, æt. 58, residing in Lincolnshire, was sent to me on June

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19th, 1891, by Dr. Hamilton, of Crowle, on account of an abdominal tumour, accompanied by persistent vomiting and rapid loss of flesh. The patient said that she had been quite well until a year and a half before admission, when she was suddenly attacked with vomiting, which had continued ever since. She had lost weight from that time until, on admission, she was worn almost to a skeleton. She had never had hæmatemesis. The vomiting took place directly after food, and was unaccompanied by nausea. At the beginning she had a stabbing pain after food in the region of the left breast, but during the six months before admission the pain had been continuous, and always worse after eating. About three months before admission she first noticed a swelling in the left hypochondrium, which had increased rapidly up to the time of admission. From the time the tumour was noticed the pain always seemed to radiate from it.

On admission the patient was extremely emaciated, and vomited everything immediately after eating. The vomit consisted simply of what she had taken, and contained free hydrochloric acid, but no sarcinæ or blood-cells could be detected microscopically. Occupying the left hypochondrium and reaching into the epigastrium was a hard nodular tumour, moving up and down with respiration, the skin being quite movable over it. A tumour could also be felt beneath the liver, which was diagnosed as a distended gall-bladder.

Cancer of the stomach was diagnosed, and the patient was fed with small quantities of Brand's essence and peptonised foods, nutrient enemata being also administered. The pain was controlled by morphia.

At first slight improvement took place, but towards the end of the month she again lost ground, and the vomiting still persisted. After consultation an exploratory incision was decided on, in order to see if the disease could be removed; but if it should prove too extensive for removal, as it was suspected it might, jejunostomy could be done.

On July 1st, 1891, the A. C. E. mixture being the anæs-

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thetic, and the skin of the abdomen having been aseptic-ised, an incision of three inches was made in the linea alba above the umbilicus, exposing the tumour, which was found to be occupying the whole of the anterior wall of the stomach, and therefore incapable of removal. There was also a distended gall-bladder, containing gall-stones.

The jejunum was then found at its fixed point on the left of the spine, and traced for about six inches downwards, at which spot a knuckle of the bowel was brought forward and fixed by loop sutures (after the parietal peritoneum and skin had been connected by a continuous suture) in the way suggested for gastrostomy by Mr. Greig Smith, and which a case of mine published in the 'British Medical Journal' for June 7th, 1890, proves to be a safe and efficient method.

A loop of silver wire was first inserted into the convex surface of the bowel at a spot where it would have to be opened. A round, large-eyed sewing needle was then threaded with twelve inches of silk of medium thickness, and passed under the peritoneal coat of the intestine in a circle about one and a half inches in diameter, the suture being made to emerge and leave five loops at equal intervals. The loops were drawn through the parietes at about one third of an inch from the margin of the wound. As each loop was drawn up a piece of No. 6 catheter was passed through it, and when all were in position the ends of the silk were drawn in and the loops tightened over the catheter, over which the two ends were then knotted; the silver wire was then fixed under the catheter, and a few sutures connected the bowel to the skin. The rest of the parietal incision was brought together by interrupted silkworm gut sutures in the ordinary way.

On the following day the patient felt quite comfortable, and was fed by the rectum entirely. This was continued until July 7th, when a small opening was made in the exposed bowel with a tenotomy knife, and a soft catheter was passed in, through which she was fed by peptonised food,

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of which she was able to take two or three pints a day. She gained flesh, and was able to return home on July 27th. Her chief trouble was that the skin around the artificial opening became irritable; but through the opening into the duodenum she continued to be able to take a fair quantity of food, which was well retained. The original disease continued to advance, and after two months she died from exhaustion.

No autopsy could be obtained.

Remarks.—With regard to the operation of jejunostomy, my experience in Case 3 would lead me to conclude that life can undoubtedly be prolonged by it in cases of cancer of the stomach too extensive for removal, and when food cannot be taken; but it seems to me that it ought to be clearly stated beforehand to the patient that relief only will occur, and that existence under the circumstances may not be particularly comfortable, although life may actually be prolonged for weeks, or even for months. An opening into the jejunum for feeding purposes is quite different from an opening into the stomach, and is not nearly so satisfactory, as the edges of the fistula tend to become irritated by bile and intestinal secretion, producing considerable discomfort.

In cancer of the pylorus, pylorectomy undoubtedly offers a chance of cure and a possibility of considerable relief, but for the operation to be successful it must be done at quite an early stage. In my second case, although the disease was to all appearances in quite an early stage, it had already involved the glands, and invaded the contiguous parts of the stomach and duodenum.

It would seem to me preferable not to be wedded to any special operation in these cases, but at the time of exploration to be guided by circumstances: for instance, where the disease is very extensive, having become fixed and having involved glands, I should prefer gastro-enterostomy to any form of pylorectomy.

Where the disease is fairly advanced, yet not too ex-

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tensive to prevent successful removal, I would suggest pylorectomy with closure of the cut ends of the stomach and duodenum, and immediate gastro-enterostomy, as in the operation performed by Mr. Lawson, Mr. Greig Smith, Dr. Bull, and Mr. Jessett.

Where, however, the disease is taken early, is not too extensive, has not involved glands, and has not fixed the pylorus to the contiguous parts, I should decidedly prefer simple pylorectomy, as in the cases I have reported; but in order to avoid the occurrence of cicatricial constriction I shall in future use a decalcified bone tube almost equal in diameter to the cut section of the duodenum, the tube being about an inch in length, with an elevated rim at each end (rather like a cotton bobbin) to prevent its displacement at too early a stage. This method occurred to me when I was operating on my first case, but as the patient had recovered and appeared so well at the time I performed the second operation, I did not feel it necessary to vary my procedure and carry out the idea, but I shall certainly carry it out in future, as I think it will give greater security at the time and prevent after contraction.

By means of the tube, and by using a continuous suture to the margins, of the openings and another continuous suture applied so as to bring the serous surfaces into apposition one third of an inch from the margins, the operation can be shortened very considerably, and on the cadaver I have performed it in quite a short time.

The diagrams shown will, I trust, serve to make plain the method of pylorectomy I suggest, and the models will, I trust, further illustrate my suggestions.

With a little modification I am employing the same tube in gastro-enterostomy, in enterectomy, and in intestinal anastomosis.

Addendum.

The modification of pylorectomy by the employment of a cotton-reel shaped tube, though simple, is difficult to

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explain without showing the tubes and illustrating their use by diagrams and models; I hope, however, that by their help I can make clear how the operation is done.

The tubes, made of decalcified bone, are of various sizes, and secure an immediate communication between the two apposed hollow viscera. The raised ends of the tube prevent the appliance slipping either into the stomach or duodenum until union is accomplished between them. Of the two continuous sutures, one is marginal, and when drawn tight causes the edges of the openings in the stomach and duodenum to be firmly applied to the tube, thus preventing extravasation of visceral contents.

The second continuous suture, placed one third or half an inch from the margin of the opening, takes up peritoneum only, and secures peritoneal apposition for fully one third of an inch from the visceral opening all round. No other sutures are required, hence the operation can be quickly performed, especially after a little practice on models or on the cadaver.

The tube can be varied in size according to the diameter of the cut section of the bowel. By securing a continuous mucous surface through the opening, a narrowing of the artificial channel will not be likely to occur; moreover, the communication between any two viscera may be made as large or as small as required, according to the size of the tube employed. The method is equally available for pylorectomy, for end-to-end enterorrhaphy after enterectomy, for intestinal anastomosis, for gastro-enterostomy, for lateral implantation as in ileo-colostomy, or for cholecystenterostomy. Last, but not least, after practice on models made of wash-leather and on the cadaver, I have proved its safety in actual practice in a case of a man aged sixty-five, suffering from stricture of the ascending colon producing intestinal obstruction, where I short-circuited the obstruction by connecting the lower end of the ileum to the colon by means of the oval modification of the cotton-reel shaped bone tube, the patient making an uninterrupted recovery, and having his bowels moved

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regularly by the rectum daily subsequent to the second day after operation. I have to thank Dr. McGregor Young for his kindness in making me the diagrams, and Messrs. Maw and Thompson for the admirable manner in which they have manufactured the tubes.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 16.)

DESCRIPTION OF PLATE VI.

Two Cases of Pylorectomy and One of Jejunostomy (Mr. A. W. Mayo Robson, F.R.C.S.).

Fig. 1.—The *continuous serous suture* is applied, a third to half an inch from the margins of the openings in the viscera, first posteriorly. The ends are left long, with the needle still threaded, in order to complete the suture after the tube has been introduced and the marginal suture completed.

Fig. 2.—The *continuous marginal suture* is applied first around the posterior margins of the openings in front of the *serous sutures* Both ends of the *suture* are left long, the needle being threaded still at one end, to complete the closure as soon as the *bone tube* is in position.

Fig. 3.—The posterior portions of the *serous* and *marginal suture* already inserted having been drawn tight, the *bone tube* is placed *in situ*, and the *continuous marginal suture* is proceeded with in front of the tube.

Fig. 4.—The *continuous marginal suture* having been completed, it is drawn tight and tied, thus making the margins of the visceral openings embrace the tube all round, and effectually closing the viscera. The *serous suture* is then continued round in front, a third to half an inch from the margins. (Above, the *serous suture* is shown *in situ* before being drawn tight.)

Fig. 5.—The *serous suture* is then tightened and cut off short, leaving a third to half an inch of serous surfaces in apposition. (The sutures are buried and cannot be seen. The tube is shown in dotted lines.)

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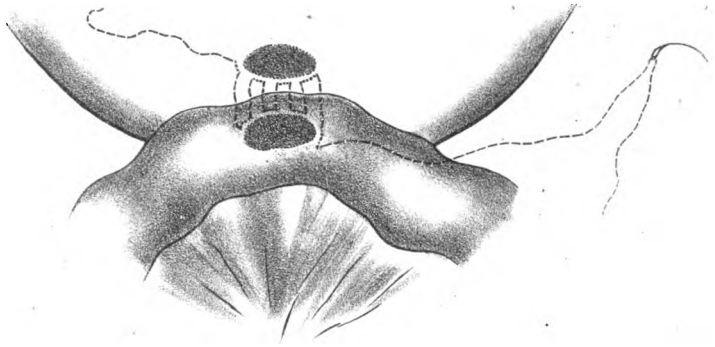


Fig. 1.

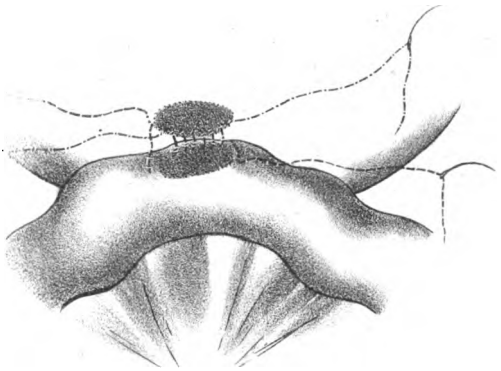
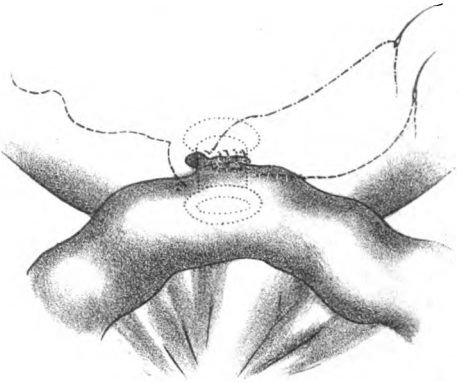


Fig. 2.



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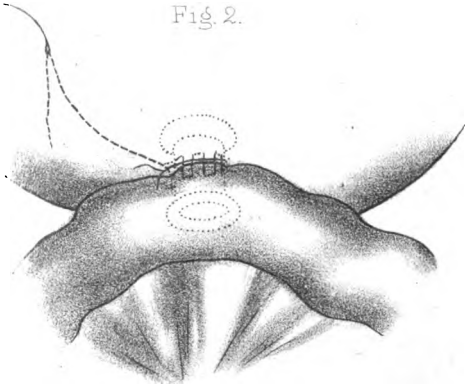

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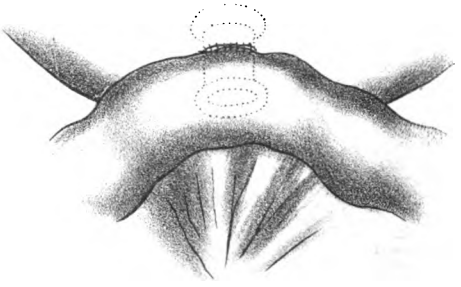

Fig. 5.

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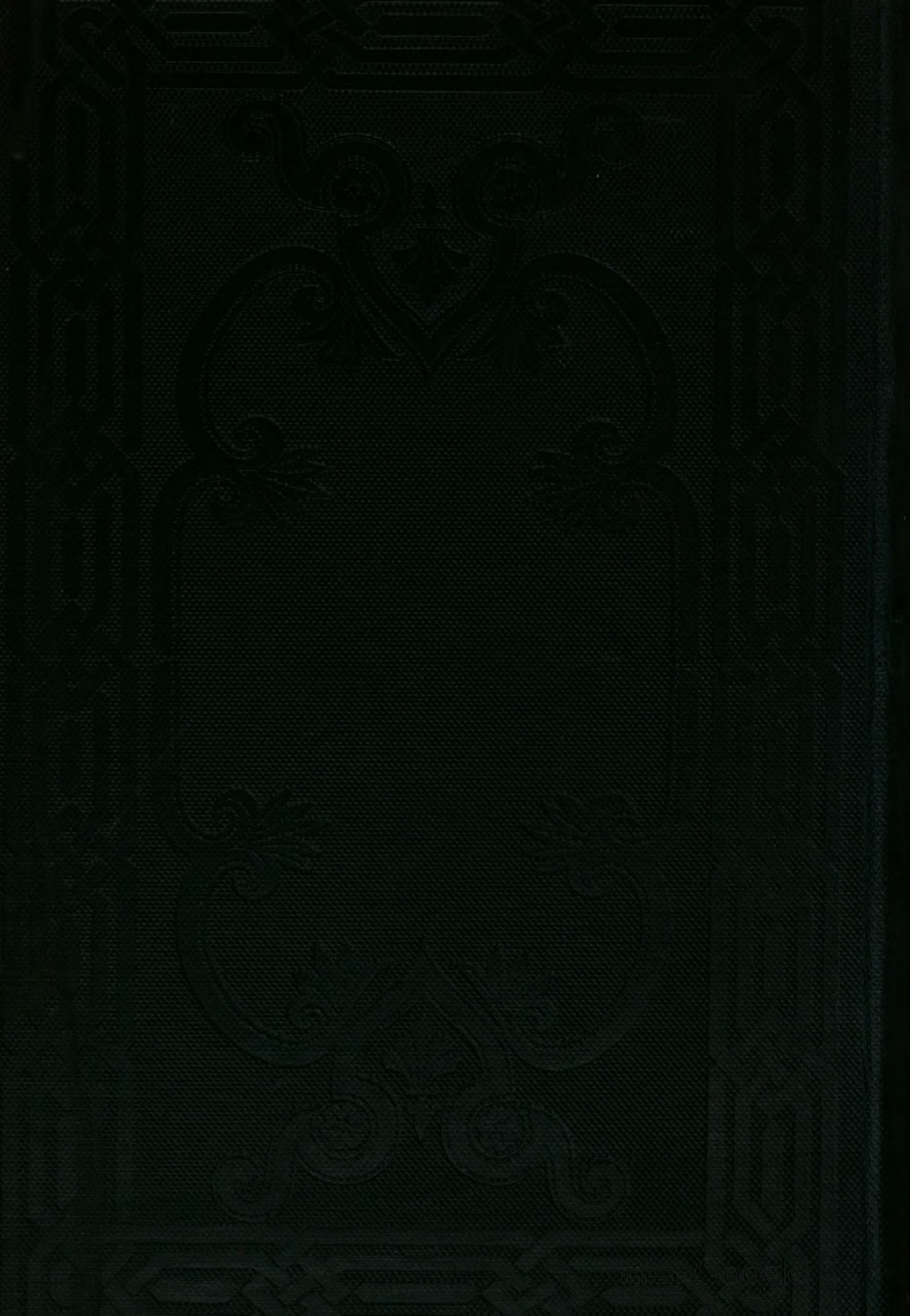
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